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VOL. XLII SEPTEMBER, 1933 No. 3

Annals of Otology, Rhinology and Laryngology

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PUBLISHED QUARTERLY
BY THE ANNALS PUBLISHING CO.
7200 WYDOWN BLVD.
ST. LOUIS, MO., U. S. A.

SUBSCRIPTION PRICE, $6.00 PER ANNUM, IN ADVANCE

Subscription price in Canada, $6.40.
Subscriptions in other countries of the Postal Union, $6.80.
Entered at the Postoffice, St. Louis, Mo., as Second-class Matter.

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OF
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AND
LARYNGOLOGY
INCORPORATING THE INDEX OF OTOLARYNGOLOGY.

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XLIX.

THE INFLUENCE OF SIR FELIX SEMON ON THE DEVELOPMENT OF LARYNGOLOGY.*

IRA FRANK, M. D.,

CHICAGO.

The modern specialty of laryngology is only about half a century old, yet diseases of the larynx were recognized in ancient medicine, and laryngologic procedures were practiced by the Egyptian and Grecian physicians. Those interested in the historical phase of diseases of the upper respiratory tract will find a paper on this subject by Milligan (1922), who gives a detailed account of the evolution of this department of medicine. The beginning of laryngology as a distinct branch of medicine in the United States had a checkered and sporadic career.

The book entitled "Growths and Tumors of the Throat" (1817) by Dr. Cheesman of New York, gave a new impetus to laryngologic inquiry and stimulated the thought of physicians along this line. Dr. Cheesman's book was a pioneer effort and was the first of its kind in American literature. Dr. Horace Green's "Treatise on Bronchitis" (1846) excited great interest and was

*Read at a meeting of the Chicago Laryngological and Otological Society, April 3, 1933.

I am indebted for the loan of these pictures, menu cards and letters, to Dr. Robert Sonnenschein, who inherited them from the late Dr. Emil Meyer and exhibited them after the reading of this paper.

Fig. 1. Sir Felix Semon in full regalia. Photograph presented to Mrs. Emil Mayer in memory of her week in London, July, 1907.

an epoch making publication from the standpoint of laryngologic literature. He described catheterization of the larynx and proved that a foreign body could be introduced into the larynx, even below the vocal cords, without producing suffocation.

Intralaryngeal surgery had been practiced to some extent before the invention of the laryngoscope by Manuel Garcia in 1858, but it is obvious that the use of this important instrument enabled physicians to make more accurate diagnosis and to exercise greater skill and technic in performing intralaryngeal manipulations.

The New York Laryngological Society is the oldest in this country. It was founded by Clinton Wagner in 1873. This was merged with the Section on Laryngology and Rhinology of the New York Academy of Medicine in 1885. The American Laryngological Association was organized in 1878. These dates are important because at this very period the diseases of the larynx were attracting the scientific attention of European physicians. Important contributions were being made concerning the structure and function of the throat, and the diseases of this region were being studied on the basis of inflammatory, neoplastic and degenerative changes.

The development of laryngology in this country is largely indebted to the stimulating influence of Sir Felix Semon, whose career and attainments I shall briefly sketch. He was born at Danzig in 1849 and had the benefit of a thorough education. His father was a wealthy broker, though he too ebbed and surged with the tide and occasionally suffered the defeat of men who abide in the marketplace. Notwithstanding this, young Semon entered the University of Heidelberg in the summer term of 1868. Here he came under the influence of such great teachers as Kirchoff the Physicist and Bunsen the Chemist. He speaks of Professor Nuhn the Anatomist, whom he recalls for his dry and uninteresting lectures and also because he most frequently lectured to vacant chairs. He also mentions the fact that he attended the lectures of the great Helmholtz. Like many another man, the course of his career was changed by an accident which occurred while horseback riding. Semon sustained a fracture of the lower portion of his femur. This prevented his return to Heidelberg.

and after his recovery he matriculated at Berlin for the second half of the winter term of 1868-69. His medical course was interrupted by the outbreak of the Franco-Prussian War. He enlisted in the cavalry service and remained on duty until the end of the war. After his discharge from military service he resumed his medical studies. The most distinguishing episode he records in his autobiography about his war experience was the fact that the regimental band, when passing through the Brandenburg gate before being mustered out, played the St. Quentin march, which he composed during his period of enlistment. In discussing in retrospect his student career at Berlin, he admits that his preliminary studies did not interest him and he looked upon them as "mere examination stuff." In his later life he regretted that he had neglected his anatomic and physiologic studies and was frequently under the necessity of intensive studies and reviews during his later active professional life.

He acknowledges his great indebtedness to his teacher in pathology, who was no other than the famous Rudolph Virchow. To the great clinician Traube he expressed his gratitude for having taught him to observe exactly and minutely and to interpret with judgment and discretion. He evidently thought that Traube had a great influence on his subsequent mode of thought and action. He held him in highest esteem. After his graduation he considered his future and career. The idea of being a general practitioner in Berlin was repellant to him, and he also acknowledges his inferiority complex, which so often exists in the consciousness of sensitive and intellectual persons of Semitic origin. That feeling pursued him like a specter through his formative period and probably through his whole life, and some of his behavior may be interpreted as defense reactions.

At the suggestion of his parents, he left Berlin for postgraduate work in Vienna, Paris and London. Before leaving Berlin he consulted his friend Professor Traube, who gave him very useful and sensible advice. In 1874 Vienna was still under the influence of the modern masters, such as Skoda, Oppolzer, Rokitansky, Helbre and others. Some of the active teachers of that period were Billroth, Bamberger, Schroetter, Politzer, Kaposi and Stoerk. In spite of this galaxy of brilliant teachers, Semon was

not so keen about Vienna. Whether this antipathy was due to his individual personality or to a nationalistic prejudice it is impossible for me to say. He refers with sarcastic darts to Gruber and Politzer and mentions, in passing, Stoerk, Schnitzler and Schroetter, and finally pays a passing tribute to Politzer and Otto Chiari, whom he terms the most prominent successors of the earliest Vienna laryngologists.

He devoted no little time and thought to the musical circles of Vienna, and he mentions by name the most distinguished personalities of this period, the most prominent of whom is Johannes Brahms. He admired his musical ability more than his personality, for Semon says in his autobiography, "Nothing made a greater impression on me than the contrast between the master's art and his personality." He says his main characteristic was a rare sarcasm in speech and thought, but we are more interested in Semon's medical than in his musical career, though it is interesting to study a great personality from the standpoints of his lights and shadows. The love of music and the ability to skillfully perform on musical instruments is a component part of man's cultural nature. In accordance with his original program, he prepared to leave Vienna for London at the end of the winter term of 1874 and 1875. He traveled by easy stages, visiting Venice, Trieste and the other Italian cities, stopping at Paris and arriving at London. After he had overcome the language difficulties he visited the various hospitals and at first was attracted to St. Thomas'. Here he heard Murchison, who lectured on clinical medicine, particularly on the fevers; also Richard Liebreich, a German, who had charge of the eye and ear department. He was also welcomed by Drs. Bristowe and Ord. After a short time he became interested in the Hospital for Diseases of the Throat, of which Sir Morell Mackenzie, Great Britain's most famous laryngologist, was chief. Semon was impressed by the vast amount of material of the throat hospital. He said that the number of patients seen in Mackenzie's hospital exceeded the number seen in Schroetter's, Stoerk's and Schnitzler's departments combined. Mackenzie's personality and skill appealed to this young man. He spent most of his time at the hospital, and in the end a friendly relationship became mutual, and Mackenzie offered him

Fig. 2. Sir Felix and Lady Semon with Dr. Emil Mayer before Grant's Tomb, Riverside Drive.

a position as clinical assistant. Semon was helpful to Mackenzie in translating and abstracting from the German laryngologic literature, and this was particularly helpful at this time to Mackenzie because he was writing a textbook on the diseases of the throat. At the same time Semon undertook to translate this book into German. In 1876 the time had arrived for Semon to select a location for permanent practice of his chosen specialty. He dreaded to return to Berlin, where the specter of antisemitism still haunted him. In order to make a decision, it was necessary to have the consent of his parents, but more especially to have the approval of Mackenzie. Mackenzie assented readily, though his parents vehemently objected. After some delay, he received the half-hearted consent of his parents. On passing his British medical examination, he was advised by Mackenzie to obtain membership in the Royal Medical and Chirurgical Society, but it is interesting to note the state of the British medical mind at this period.

Mackenzie advised him not to mention that he was connected with the throat hospital, because, as Mackenzie said, "Laryngology is not popular with the medical profession and British medicine is extremely conservative and hates specialism." Nevertheless, Semon was elected to the Royal Medical and Chirurgical Society after this conversation. The first period of Semon's practice was not characterized by lucrative returns. In the meantime the young laryngologist was making some friends in social circles, but was more particularly attracted to musical people. He played the piano and wrote musical compositions, which increased his circle of acquaintanceship. It may be judged from what has already been said that Semon was a temperamental person. He had the happy faculty of disagreeing with and parting from his old friends. This is not always due to a personality defect. The jealousies of medical practice are too well known to dwell upon.

In Great Britain the traditions of medical practice were opposed to specialism; besides Semon was a foreigner and not an Aryan. Yet we cannot wholly excuse Semon, because we believe he was sensitive and at times quarrelsome. Semon fell out with Mackenzie and their social relationship ceased in 1881. A most unpleasant consultation in 1882 parted them in a professional way for good.

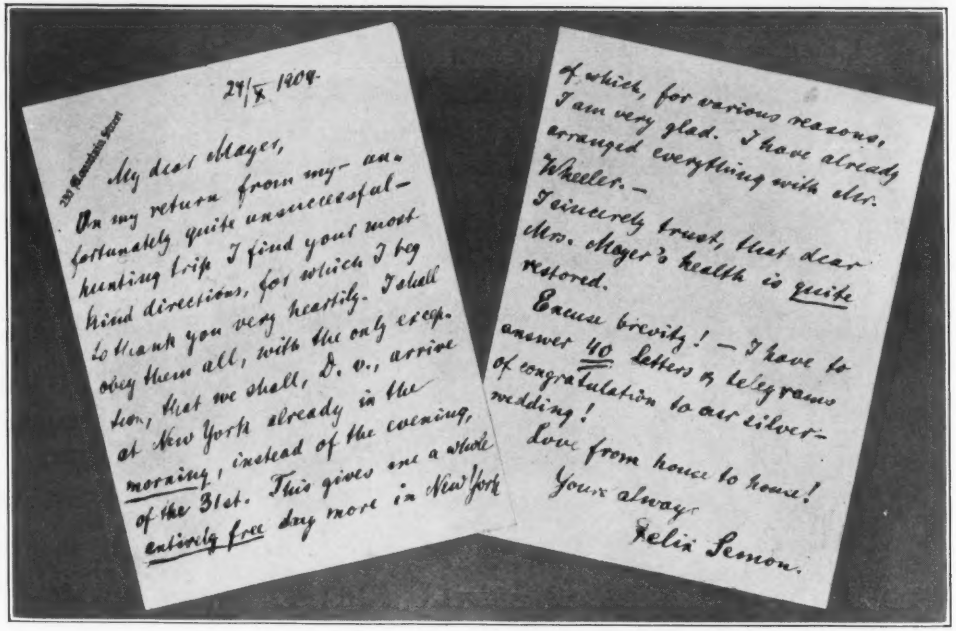

29/8 1904.
My dear Mayer,
On my return from my unfortunately quite unsuccessful hunting trip I find your most kind directions, for which I beg to thank you very heartily. I shall obey them all, with the only exception, that we shall, D. v., arrive at New York already in the morning, instead of the evening, of the 31st. This gives one a whole entirely free day more in New York
of which, for various reasons, I am very glad. I have already arranged everything with Mrs. Wheeler. —
I sincerely trust, that dear Mrs. Mayer's health is quite restored.
Excuse brevity! — I have to answer 40 letters & telegrams of congratulation to our silver-wedding!
Love from house to house!
Yours always
Felix Semon.

Fig. 3. Letter from Sir Felix Semon to Dr. Emil Mayer, dated October 24, 1904, announcing the date of his expected arrival in New York.

Enough has been said to indicate the nature and quality of the man, at least for the moment. Let us now turn aside to consider his scientific achievements, his contributions and his progress in establishing fundamental facts in laryngology.

Sir Felix Semon worked diligently and brilliantly in establishing laryngology as a special branch of medicine. He unraveled many hidden causes of diseased conditions, and he interpreted the nature of a variety of disorders so that he may be regarded as one of the pioneers in laryngology, and he made a position for it as a specialty. The influence of Semon became international. He aroused interest in the subject everywhere and stimulated research and investigation throughout the world in this branch.

Semon's real work in connection with the recognition of laryngology as a distinct specialty began with the International Medical Congress of London in 1881, where he succeeded in having laryngology recognized as such. There was at this time in England a pronounced tendency to discredit the subject and those who practiced it. In the larger hospitals throat cases were handled by an assistant surgeon or by a physician of the eye, ear, nose and throat department. The Medical Congress had arranged for separate sections for otology, ophthalmology, dermatology and odontology. Rhinology was scarcely recognized, but Semon obtained a section for laryngology.

Just fifty-one years ago, in 1882, Semon was appointed physician for diseases of the throat at St. Thomas' Hospital, one of London's largest institutions. Thus the tradition of neglect was broken; laryngology was for the first time fully recognized as a specialty, since no English hospital had previously made a special appointment in this field. In 1885 the Royal College of Physicians elected Semon a Fellow. As this was the first time such a distinction had been conferred on one confining his practice to diseases of the throat, it was an emphatic testimonial to the position at which he had now arrived.

These three British moves had decisive influence on the entire medical world. Semon was looked upon as the leading figure in the new specialty, and his founding of the *Internationales Centralblatt für Laryngologie* in 1884 gave the journal an international standing and provided a medium for special laryngologic litera-

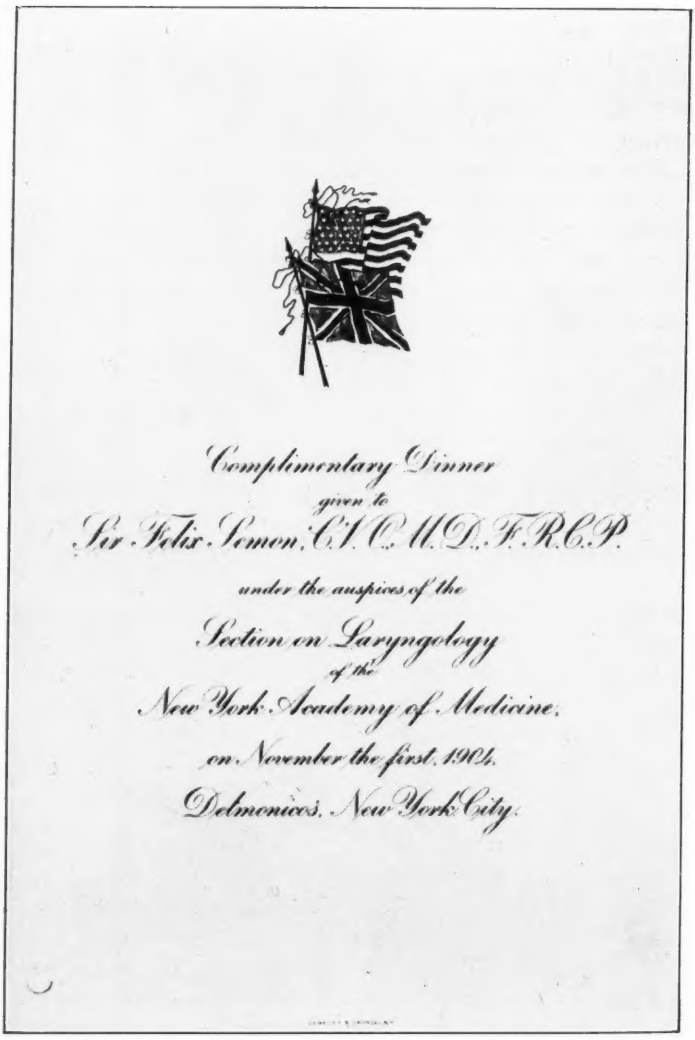


Fig. 4. Cover of menu of complimentary dinner given to Sir Felix Semon.

ture. Semon continued as its editor for twenty-five years, until 1909. As a reward for these services as they came along, Semon was elected honorary fellow of most of the newly formed laryngologic societies throughout the world, including the American Laryngological Association and the American Laryngological, Rhinological and Otological Association. Their existence was largely due to his efforts. He was knighted in England and decorated by many European and other sovereigns. His influence in laryngology was world wide, both by personal contacts and by his writings. It should be remarked that Semon did not like the division of laryngology into subsections. He did not like the union of otology and rhinology. He objected to the American view that the laryngo-otologist should extend his operative domain to the adnexa and possible complications. He preferred keeping laryngology pure and simple. Nevertheless, he was not a narrow specialist, but remembered always that he was first of all a physician, not divorced from general medicine and surgery. He made it a point to visit the medical wards regularly.

The two main subjects in laryngology with which Semon's name will always be associated are: Laryngeal paralysis and malignant disease of the larynx.

Before his time the whole subject of laryngeal paralysis was chaotic, both in didactic teaching and in textbooks. From an early period in his practice he had observed the greater frequency of abductor over adductor paralysis, and at his suggestion Mackenzie in his great work on "Diseases of the Throat and Nose" (1880), referred to this greater frequency. Semon translated this work into German and added several contributions of his own to this section.

To Semon undoubtedly belongs the credit of enunciating and proving that "In central and peripheral progressive organic lesions of the motor laryngeal nerves the abductors alone suffer or at least do so earlier and to a greater extent than the adductors." This is known as "Semon's law," which he developed in a number of papers in the English and German literature. Here are also included the occurrence of bilateral abductor paralysis in locomotor ataxia and more particularly the relation of functional to organic laryngeal paralysis. Previous to his time it was thought

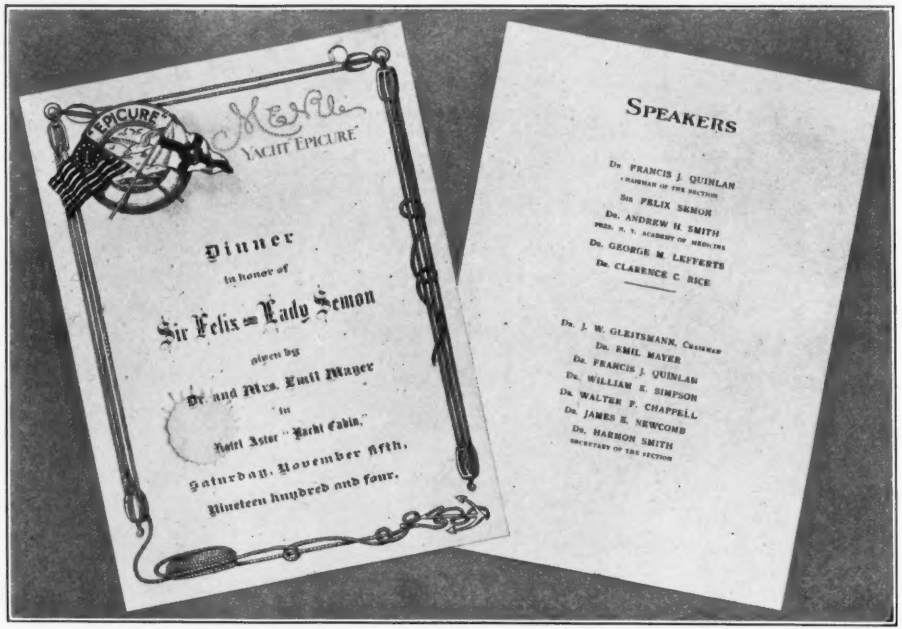


Fig. 5. Dinner in honor of Sir Felix and Lady Semon, given by Dr. and Mrs. Emil Mayer, November 5, 1904.

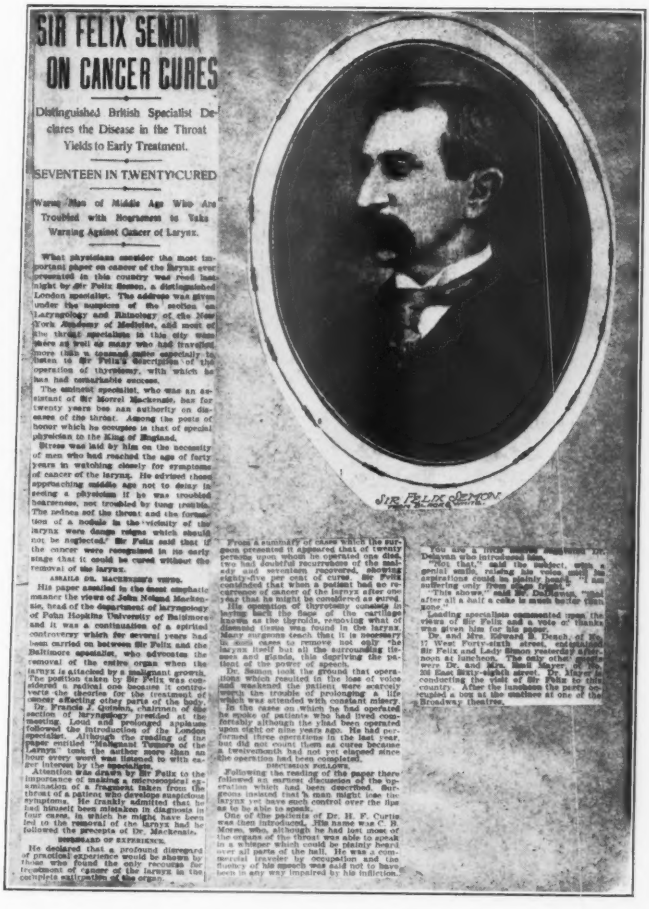
Fig. 6. Autographs of distinguished guests at a dinner given in honor of Sir Felix and Lady Semon.

to be a mere matter of chance which muscles were affected. Owing to his researches it became obvious that motor paralysis of the larynx might be central or peripheral, that the abductors and adductors might be involved, but that in organic disease the adductors were not primarily affected except in some rare cerebral cases; also that in purely functional cases the adductors alone were involved.

In association with Victor Horsley, the neurologist, Semon in 1890 published an epoch making paper which showed that it was probably impossible for a one-sided cortical lesion to produce laryngeal paralysis; that laryngeal paralysis which involves the abductors (and only very rarely the adductors as well) is due to organic disease, while motor paresis of the adductors is, in the great majority of cases, functional, more rarely, myopathic, and then commonly due to catarrh. This paper is the framework upon which our modern knowledge of motor neuroses of the larynx is based.

Semon's great contributions to laryngology, both here and in Europe, were in conection with the "laws" that bear his name, concerning the innervation of the larynx and his pioneer work in the diagnosis and treatment of laryngeal growths; his outstanding work should be mentioned on the pathology of edematous and other laryngitides and on pneumococcus infections of the throat which were previously thought to be syphilitic.

It is interesting to note here that at the International Medical Congress in 1881 there was not a single paper on the surgery of the nose, on diseases of the accessory sinuses, on the bacteriology of the air passages, on laryngofissure, or any of the throat subjects previously mentioned. But after Semon began to develop laryngology most of the special subjects dealt with by the present day otolaryngologist were commonly discussed in half a dozen or more special journals. Undoubtedly the efforts of Semon gave laryngology scientific standing and influence. By his careful research and his own masterful pen, as well as by his encouragement of others, he made important contributions to every phase of the subject—theoretical, technical and clinical—for the consideration of specialists in this field of medicine all over the world. Semon wrote not for the sake of writing or of increasing



SIR FELIX SEMON ON CANCER CURES

Distinguished British Specialist Declares the Disease in the Throat Yields to Early Treatment.

SEVENTEEN IN TWENTY CURED

Warns Men of Middle Age Who Are Troubled with Hoarseness to Take Warning Against Cancer of Larynx.

What physicians consider the most important paper on cancer of the larynx ever presented in this country was read last night by Sir Felix Semon, a distinguished London specialist. The address was given under the auspices of the section on Laryngology and Rhinology of the New York Academy of Medicine, and most of the throat specialists in this city were there as well as many who had travelled more than a thousand miles especially to listen to Sir Felix's description of the operation of thyrotomy, with which he has had remarkable success.

The eminent specialist, who was an assistant of Sir Morrel Mackenzie, has for twenty years been an authority on diseases of the throat. Among the posts of honor which he occupies is that of special physician to the King of England.

Stress was laid by him on the necessity of men who had reached the age of forty years in watching closely for symptoms of cancer of the larynx. He advised those approaching middle age not to delay in seeing a physician if he was troubled by hoarseness, not troubled by long trouble. The redness of the throat and the formation of a nodule in the vicinity of the larynx were danger signs which should not be neglected, Sir Felix said that if the cancer were recognized in its early stage that it could be cured without the removal of the larynx.

ASSAILS DR. MACKENZIE'S VIEWS.

His paper assailed to the most emphatic manner the views of John Mcleod Mackenzie, head of the department of laryngology of Johns Hopkins University of Baltimore and it was a continuation of a spirited controversy which for several years had been carried on between Sir Felix and the Baltimore specialist, who advocates the removal of the entire organ when the larynx is attacked by a malignant growth. The position taken by Sir Felix was considered a radical one because it contravenes the theories for the treatment of cancer affecting other parts of the body.

Dr. Francis J. Quinlan, chairman of the section of laryngology presided at the meeting. Loud and prolonged applause followed the introduction of the London specialist. Although the reading of the paper entitled "Malignant Tumors of the Larynx" took the author more than an hour every word was listened to with eager interest by the specialists.

Attention was drawn by Sir Felix to the importance of making a microscopical examination of a fragment taken from the throat of a patient who develops suspicious symptoms. He frankly admitted that he had himself been mistaken in diagnosis in four cases, in which he might have been led to the removal of the larynx had he followed the precepts of Dr. Mackenzie.

STANDARD OF EXPERIENCE.

He declared that a profound disregard of practical experience would be shown by those who found the only reasons for treatment of cancer of the larynx in the complete extirpation of the organ.

From a summary of cases which the surgeon presented it appeared that of twenty persons upon whom he operated one died, two had doubtful recurrences of the malady and seventeen recovered, showing eighty-five per cent of cures. Sir Felix considered that when a patient had no recurrence of cancer of the larynx after one year that he might be considered as cured.

His operation of thyrotomy consists in laying back the faces of the cartilage known as the thyroids, removing what of diseased tissue was found in the larynx. Many surgeons teach that it is necessary in such cases to remove not only the larynx itself but all the surrounding tissues and glands, this depriving the patient of the power of speech.

Dr. Semon took the ground that operations which resulted in the loss of voice and weakened the patient were scarcely worth the trouble of prolonging a life which was attended with constant misery.

In the cases on which he had operated he spoke of patients who had lived comfortably although the triad had been operated upon eight or nine years ago. He had performed three operations in the last year, but did not count them as cures because a twelvemonth had not yet elapsed since the operation had been completed.

[illegible] FOLLOWS.

Following the reading of the paper there followed an earnest discussion of the operation which had been described. Surgeons insisted that a man might lose the larynx yet have such control over the lips as to be able to speak.

One of the patients of Dr. H. F. Curtis was then introduced. His name was C. B. Moore, who, although he had lost most of the organs of the throat was able to speak in a whisper which could be plainly heard over all parts of the hall. He was a commercial traveler by occupation and the fluency of his speech was said not to have been in any way impaired by his infliction.

[illegible] Delavan who introduced him.

"Not that," said the subject, with a genial smile, raising his voice, until his aspirations could be plainly heard, "I am suffering only from high living."

This shows," said Mr. Dorlinson, "what after all a half a cake is much better than none."

Leading specialists commented upon the views of Sir Felix and a vote of thanks was given him for his paper.

Dr. and Mrs. Edward B. Dench, of No. 17 West Forty-sixth street, entertained Sir Felix and Lady Simon yesterday afternoon at luncheon. The only other guests were Dr. and Mrs. Emil Mayer, of No. 32 East Sixty-eighth street. Dr. Mayer is conducting the visit of Sir Felix to this country. After the luncheon the party occupied a box at the matinee at one of the Broadway theatres.

Fig. 7. Newspaper interview with Sir Felix Semon with reference to his paper on Cancer of the Larynx.

his reputation. He wrote because he felt that he had a message to deliver; hence his papers, though ponderous, were always new and interesting and elicited thought and controversy which were the life of the young specialty. His articles dealt more with the scientific aspects of laryngology than with the purely clinical and therapeutic sides; but he did not hesitate to draw on his large clinical experiences in illustrating his views and enforcing critical judgment.

Semon was probably the first, in 1883, to associate the absence or removal of the thyroid gland with cretinism and myxedema and the cachexia strumipriva of Kocher. Horsley's experiments with sheep confirmed Semon's conclusions and laid the foundation of our present organotherapy. The two men were lifelong friends and collaborators.

The personality and social experiences of Semon are worth dwelling upon again. He was a man of high culture, acquired not too rigidly in the educational opportunities and classic ideals of Imperial Germany and Victorian England from 1850 on. When seven years old his father let him stay up one night to see the magnificent comet of 1856, in company with the greatest scientist of that period, Alexander von Humboldt. Both were vivid memories sixty years after. At the age of 10 he had read many of the German, French and English masterpieces. These three languages he had at his command all his life. Familiar with ancient classics, he embellished his professional papers and addresses with quotations in the fashion of the day. He considered himself romantic in his taste in current literature; probably this compensated for his high scientific achievements in their exacting technical form.

His most distinctive artistic gift was music. Very early he was put to piano practice under a superior teacher, whose own training was derived from a disciple of Beethoven himself. At 8, Semon played extremely well. In England, while writing scientific papers, he was also setting most of Paul Heyse's poems to music. His wife was a beautiful contralto singer, one of whose triumphs was to sing for Liszt in his own home his setting of "Kennst Du das Land," with Liszt himself accompanying her. Rubenstein was very often in the Semon home. Later on, at the artless request

of Semon's little boy, Paderewski played two enchanted hours for the small listener on a low stool, a distinguished company in the background sharing the spell. Not only musicians famous in the London of that time, but artists, actors and literary folk thronged to enjoy the genuine and not elaborate Semon hospitality.

Fig. 8. Caricature by Spy, presented to Dr. and Mrs. Emil Mayer, 1902.

Like most first-class medical men, Semon was a fine organizer. It was hard for him to put up with the pitiful equipment and assistance of his early English clinics. As soon as possible he improved them. He was careful and scrupulous in business dealings. In his diary are recorded painstakingly his income, year by year, and the number of his patients. He never allowed anything to interfere with his duty to his patients. Once when King

Edward VII, whose physician he was for a long time, sent for him to call at nine to treat a royal sore throat, Semon sent back word that he had a most unusual operation, the second only of its kind, to perform at 8:00 o'clock on a much frightened patient; would his Majesty not permit him to call a little later in the morning? For many years Sir Felix attended royalty from Queen Victoria's family on down to even a third generation. As he became an unusually keen and successful sportsman after coming to live in England, he was frequently a guest at the hunting boxes of royalty and the English nobility.

On his retirement, in 1910, after thirty-three years of active practice, friends all over the world showed their appreciation of his services to laryngology by subscribing to the endowment of a permanent lectureship, the Semon Lecture, which is given annually. At the London banquet at which this announcement was made in his honor, all who were foremost and brilliant in political, professional, social and artistic society were present to show their high regard of the man and his work.

In 1910 he edited and published his collected works in two volumes: "Forschungen und Erfahrungen, 1880-1910, von Professor Dr. Sir Felix Semon, K. C. V. O." (Two volumes, Berlin, Hirschwald, 1912.)

In 1926 appeared his autobiography, a complete life record, consisting of 349 large pages. He began to prepare it in 1912, three years after his retirement and after a ten months' journey around the world. His clear memory for facts, and the collection of letters and documents accumulated through his active years, also diaries kept over considerable periods of time, were his working material. After his death, in 1921, his son and able assistants brought it to completion.

To realize fully his success, we must remember who and what he was when he first appeared among that traditionally conservative class, the British medical profession. Semon was a stranger among them, an alien, who advocated and practiced a specialty which was under a cloud and the recognition of which he accomplished against the opposition of some of the highest authorities in the profession and the longest established medical institutions in the world.

Although we in America had less direct contact with Semon than his confreres of the continent, his work influenced the rise and growth of laryngology in America. American laryngologists contributed to his *Centralblatt* frequently. Dr. Lefferts, of New York City, was his devoted friend for forty years. Semon spoke of him as "My most faithful and indefatigable collaborator in the *Centralblatt,*" to which in the twenty-five years of Semon's editorship Lefferts had furnished more than 17,000 reports.

Sir Felix paid one visit to America, when he was invited to address the Congress of Arts and Sciences at the St. Louis Exposition in 1904. He visited and was feted in many cities along the way, meeting most of the laryngologists and many leading physicians. Almost inevitably his address at the Congress was on "The Relations of Laryngology and Otology to Other Arts and Sciences." Who in the entire world was better fitted to expound these relations? From 1877 on he had been differentiating them out of general medicine into worthy and specialized independence.

The test of wartime loyalty is cruel and exacting to a sensitive mind. What one owes to his mother country as compared to his debt to the land of his choice and adoption is not always easy to decide. Semon, though accused and suspected, left no doubt where he stood. He gave allegiance and loyal support to the country which adopted him in her hospitable embrace. He denounced in public print the cruelties and barbarities of England's wartime enemy. He sent two sons to the defense of his adopted country. In Germany he was expelled from the medical associations. His name was removed from his beloved *Centralblatt,* and in spite of suspicion at home and prejudices abroad, he stood courageously for the position he had so honorably assumed, and eventually won the respect and admiration of his colleagues.

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L.

THE SPREAD AND PHAGOCYTOSIS OF PARTICULATE MATTER IN NASAL MUCOUS MEMBRANE OF THE RABBIT.*

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ST. LOUIS.

Our knowledge of the immunity of the tissues of the nose and paranasal sinuses has been most inaccurate, and not until the last few years have investigators concerned themselves with the study of the changes which actually take place within these tissues at the invasive stage of an infection. This knowledge is essential, since these areas are presumed to be the portals of entry of so many local and systemic infections. This presumption, though generally accepted, is but meagerly substantiated by the small amount of data available in the literature concerning the penetration of infection through the epithelium, the manner in which the infection spreads in the subepithelial tissues, and the specific phagocytic cells which are active in the mucous membrane of the nose and nasal accessory sinuses in limiting the infection.

Several years ago we[1] studied the reactions of the tissues of the turbinates and the sphenoid and ethmoid sinuses in chronic infections, and noted certain changes in the epithelium, as well as a marked increase in connective tissue in the tunica propria, in the bone and about the blood vessels. This illustrated that the principal reaction to infection occurred in the connective tissue elements. In a later investigation,[2] from our examinations of the mucosa of the eustachian tube, middle ear and mastoid cells in mastoiditis, we concluded that the greater evidence of infection was apparent about the vessels of the subepithelial areolar tissue as judged by the marked perivascular cellular infiltration.

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*Read before the American Laryngological, Rhinological and Otological Society, Chicago, Ill., June, 1933.

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Mullin,[3] in 1919, by injecting India ink into the antrums of rabbits and scarifying the mucosa, succeeded in tracing the carbon deposits by way of the submaxillary and jugular nodes, the lymphatic ducts, the great veins and the right side of the heart to the lungs. His conclusion was that the carbon particles had gained entrance through the lymphatics of the antral mucosa.

Knowlton and McGregor,[4 5] from their very thorough and exhaustive studies on the regeneration of the antral mucosa in humans and animals, concluded that mucosa regenerated well despite infection, which seems paradoxical in the light of our concept of the reaction of tissues to infection, though it makes us realize that there are many influences in tissue reactions of which we know but little. This very uncertainty has prompted us to pursue this series of investigations, with the hope of adding at least a few facts to our present comparatively meagre knowledge.

Ciliary action, as the first line of defense of the nasal and sinus mucous membranes, is being carefully studied by many investigators. Hilding's[6 7] latest contributions have been most enlightening. He has shown that there is an unbroken protective layer of mucus that overlies the surface of the nasal mucous membranes that is continuous with that in the pharynx and esophagus, and that this layer is changed about once every ten minutes in the posterior two-thirds of its extent, and about once an hour in its anterior third. It is motivated by ciliary action and traction, and certain factors accelerate and diminish the speed of the cilia. India ink dropped upon the membranes of adult human beings was seen to be enmeshed and carried away by this mucinous coat. Foreign particles are thus prevented from penetrating the epithelium, and should the ciliary stream stop, the mucinous coat be altered or removed, or there occur a break in the epithelium, the optimum condition for penetration into the mucosa is presumably established.

Certain responses of the epithelium to carbon particles in other parts of the respiratory tract have been observed by Ropes, Henke, Lenart and others.

Ropes[8] demonstrated that the ciliated epithelial cells of the trachea and bronchi are phagocytic for carbon dust particles but

not for carbon in suspension. F. Henke[9] found carbon particles intercellularly and intracellularly in the epithelium of the tonsils of humans and cats, several hours after the injection of the posterior ends of the turbinates and the septum. Lenart[10] has likewise described similar findings in the tonsil of the dog after injecting the posterior end of the superior turbinate with India ink solution. He also found carbon particles in the mucous glands, in the alveoli, and between the alveolar cells of the turbinal mucosa.

PHAGOCYTOSIS.

Downey[11] described phagocytosis as the process which quickly cleans out the particles of injected suspensions from the fluids of the body, and concluded "that phagocytosis is a physiologic process which is not confined to any one type of cell, and that the material to be phagocytosed which under ordinary conditions is taken up by a certain type of cell may, under slightly different conditions, be taken up by cells which genetically and structurally are quite distinct from the first type." The principal phagocytic cells are the histiocytes, the fibroblasts, the monocytes, the polymorphonuclear leucocytes and the lymphocytes.

We do not purpose to enter into a discussion of the origin of the histiocyte, as that is an histologic problem too lengthy and far too technical for this presentation. Cowdry,[12] Maximow,[13] Aschoff and Kiyono,[14] Fenton and Larsell[15] and others who have devoted much time to this study agree that it is a cell of connective tissue origin, particularly differentiated to take up and store particulate matter. Sabin[16] classifies it as an extravascular cell from the outer wall of the vascular endothelium, the same as the resting and active wandering cell of Maximow. It is variously spoken of as the clasmatocyte and megalocyte.

The appearance of the histiocyte in our tissues stained with hematoxylin and eosin was practically identical with that of the free cell, as described by Dameshek.[17] The protoplasm and cell outlines were very indistinct, though the irregularities of the latter could be seen. In shape it was roughly triangular, ovoid or irregular due to its pseudopodial projections. It stained very pale grayish blue or pale purple. The nucleus was round, elliptical

or slightly indented, and from one-third to three-fourths the size of the cell. It stained a deep cherry red color, and the outline or nuclear membrane, the course striations and nucleolar particles a deeper shade of reddish purple. The carbon particles were characteristically arranged in a semicircle about the nucleus, with an occasional particle nearer the periphery, though some cells were completely filled. (Fig. 9.) Many carbon particles appeared to be attached to the cell membrane. This is easily conceivable when we realize that the cell membrane is extremely delicate and fragile, is moist and has adhesive properties, so that the first step towards an ingestion of a foreign particle is the attachment to the cell membrane, and then either a penetration of the membrane and ingestion of the particle, or its incorporation by the pseudopods of the histiocyte.

Downey[18 19] has found polymorphonuclear leucocytes phagocytic for trypanblau and lithium carmine within from one and one-half to two hours after the intravenous injection, or when the cells and the dye are in contact for a sufficient length of time, viz., twenty-four hours in a ligatured femoral vessel. However, he did not find the granules in the polymophonuclear leucocytes after the subcutaneous injection of the dye up to the sixth hour or after the eighteenth hour, and very few within the polymorphonuclear leucocytes at the twenty-fourth hour. He states that lymphocytes are phagocytic for dye granules when they have reached the stage of growth that they have sufficient cytoplasm to ingest the granules.

PURPOSE.

The object of these experiments was twofold. First—To ascertain whether or not inert microscopic particles would penetrate the normal intact epithelium of the nose and the antrums of rabbits if kept in contact for a sufficient length of time. Second—To study the manner in which the particulate matter spread through the subepithelial tissue, when injected directly into it, and the reactions of the cells to the foreign particles.

The factors which influence penetration through epithelium or into cells are physical, chemical, electrical and osmotic tension. These may act singly or their actions may overlap, being modified

by the type of substance seeking to gain entrance—that is, whether it has physical, chemical, electrical or osmotic properties.

I shall not attempt to delve into the question of the osmotic and chemical relationship of tissue cells and body fluids to bacterial invasion, as many able workers in the field of biochemistry have more aptly explained these phenomena; nor shall I but mention the electrical potential of tissues or living organisms, a phenomenon about which so little is known, though some excellent work is in its inception.[20] These are undeniably important factors, and much work is yet to be done before we shall fully appreciate how important they are.

In order to better study the actions of tissue cells and body fluids in the nasal and sinus mucosas, without the conflicts of the biologic activities of living organisms, I chose as a potential invader inert particulate matter in the form of India ink, as simulating as nearly as possible the substance of bacterial bodies in a readily identifiable form.

PROCEDURE.

Rabbit 1.—India ink diluted 1:20 with sterile distilled water was sprayed into each nostril of an apparently uninfected rabbit three times at ten-minute intervals, the intention being to keep the ink in contact with the mucosa constantly for thirty minutes, causing as little trauma as possible by spraying only after the ten-minute interval necessary to cleanse the previous ink. (Hilding[6 7].) The rabbit was killed by ether inhalation after six hours. The middle turbinates and adjacent antrums together with the inferior turbinates were removed with the respective lateral walls. The four pieces were immediately placed in Zenker's solution for fixation, decalcified and embedded in paraffin. The sections were cut serially at four micra in various planes and stained with hematoxylin and eosin, except every fifth section, which was stained with eosin only, to be studied as a tracer. This technic was followed in the preparation of all specimens.

Rabbit 2.—The diluted India ink (0.5 cc.) was injected into each antrum through the lateral membranous windows, without traumatizing the mucosa any more than the needle puncture necessitated. Killed in six hours by ether.

Rabbit 3.—The diluted India ink solution (0.5 cc.) was injected subepithelially into the anterior end of the left inferior turbinate, and the same amount into the anterior end of the right middle turbinate. No nasal infection present. Killed after six hours with ether.

Rabbit 4.—The diluted India ink solution (0.5 cc) was injected into the anterior ends of each of the four turbinates of a normal rabbit. After twenty-four hours the rabbit was killed by injecting air into the heart.

Rabbit 5.—The anterior end of each turbinate of a rabbit with purulent secretion in both sides of the nose was injected with 0.5 cc. of the diluted India ink solution, and the rabbit killed by injecting air into the heart after twenty-four hours.

Rabbit 6.—The India ink was diluted 1:20 with d'Herelle's bacte-rhino-phage,[21] a filtrate from broth cultures of streptococcus, staphylococcus, pneumobacillus, B. proteus, B. para-coli and B. pyocyaneus, in suspension in sterile broth. A normal rabbit was selected and the anterior end of each of the turbinates injected with 0.5 cc. of this solution, and after twenty-four hours the rabbit was killed by injecting air into the heart.

FINDINGS.

Rabbit 1.—The epithelial layer, both the ciliated columnar and squamous, throughout the turbinates and the antrums was intact and there was evidence of residual India ink on the surface of the inferior turbinates but none in the antrums. There were no carbon particles found within the epithelial cells, penetrating the epithelium, or in the subepithelial spaces or cells, nor was there any evidence of phagocytosis by the ciliated or squamous epithelium. (Fig. 1.)

Rabbit 2.—Antral cavities contained large amounts of exudate composed of viable polymorphonuclear leucocytes, many monocytes and young connective tissue cells and a small number of histiocytes. The carbon particles were seen within the protoplasm of the monocytes, young connective tissue cells and histiocytes, and also free, but not within the polymorphonuclear leucocytes. (Fig. 3.) The ciliated columnar epithelium was intact in most

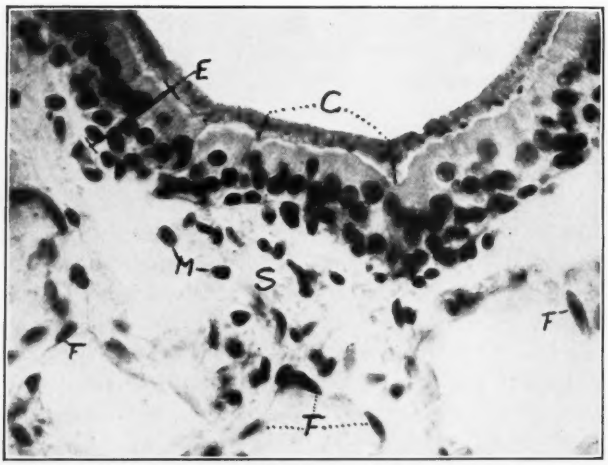

Fig. 1. Inferior turbinate. Rabbit No. 1. Carbon in suspension (C) in contact with ciliated columnar epithelium (E). No penetration into epithelium or stroma (S). (F) Fibroblasts. (M) Monocytes. Hematoxylin and eosin stain. (High power magnification.)

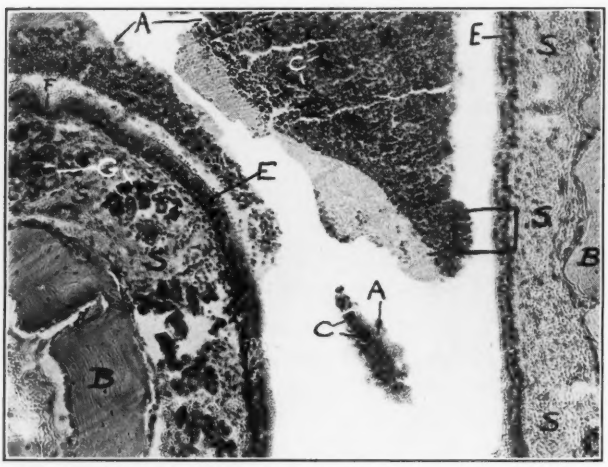

Fig. 2. Ostium of maxillary antrum. Rabbit No. 2. Exudate (A) composed of polymorphonuclear leucocytes, histiocytes, monocytes and carbon particles (C). Carbon within histiocytes and monocytes only. Ciliated columnar epithelium (E) intact, and broken (F) carbon particles not penetrating. Stroma (S) containing glands (G) and fibroblasts; no carbon. (B) Bone. Hematoxylin and eosin stain. (Low power magnification.)

places, had sloughed away down to the basal cells in others, and appeared to have broken off in blocks in others with the ciliated surfaces in contact with the cellular masses of the cavity, suggesting a continued activity after separation from the epithelial layer. There were no carbon particles found within the epithelial cells, attached or free, none penetrating the intact or the sloughed epithelium and none within the subepithelial layer.

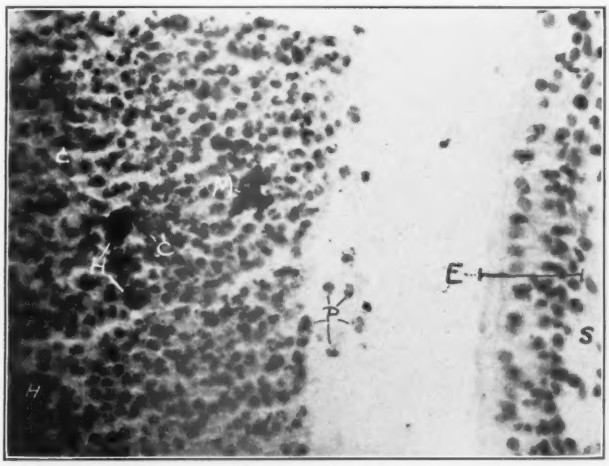

Fig. 3. Maxillary antrum, Rabbit No. 2. The majority of the cells are seen to be polymorphonuclear leucocytes (P) not containing carbon. Occasional histiocyte (H) and monocyte (M) loaded with carbon. Extracellular carbon particles (C) scattered throughout mass of cells not penetrating ciliated columnar epithelium (E) into stroma (S). Hematoxylin and eosin stain. (High power magnification.) (Outlined in Fig. 2.)

The second phase of the problem was concerned with the actual disposition of the carbon particles when introduced directly into the subepithelial tissues—that is, through what channels and in what direction the carbon particles spread, and the specific cells which phagocytosed them.

Rabbit 3.—We wished to ascertain the direction that the carbon particles would take in each injected turbinate, and whether they would spread to the uninjected turbinates on the respective sides of the nose or into the antrums.

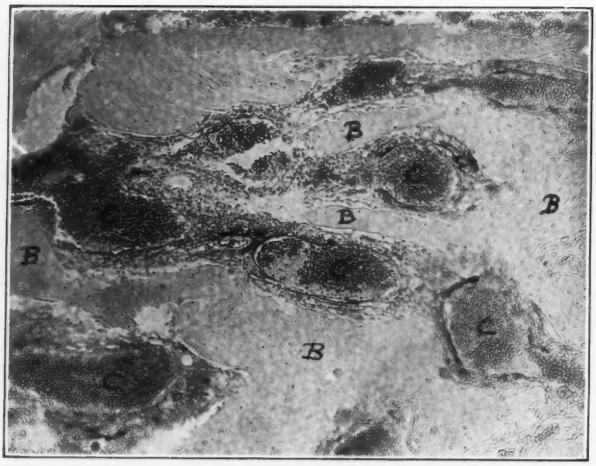

Fig. 4. Free carbon (black) in perivascular capillary lymphatics in bone spaces. (C) Blood capillaries. (B) Bone. Eosin stain. (Low power magnification.)

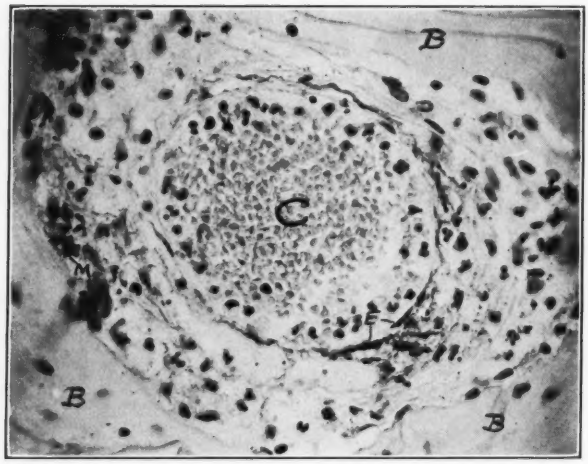

Fig. 5. Blood capillary (C) in stroma of bone space. Perivascular carbon showing as black wavy striations in tissue spaces and lymphatic capillaries. Carbon also appears within monocytes (M) and as black granules throughout stroma, none seen in blood capillary; leucocytes around periphery of lumen. (T) Endothelial cells containing carbon. (B) Bone. Hematoxylin and eosin stain. (High power magnification.)

The carbon particles were chiefly found free in the tissue spaces, where they occurred in two characteristic arrangements, one as an irregular distribution, and another as fine hair-like, solid and interrupted, strands or streams connecting definite lymphatic capillaries. (Fig. 4.) They were in the larger and smaller lymphatics, especially in the perivascular zones (Figs. 4 and 5.) and immediately underneath the epithelium. (Fig. 6.) None were seen within the blood vessels. (Fig. 5.) The larger amounts appeared to be in the posterior halves of the injected turbinates,

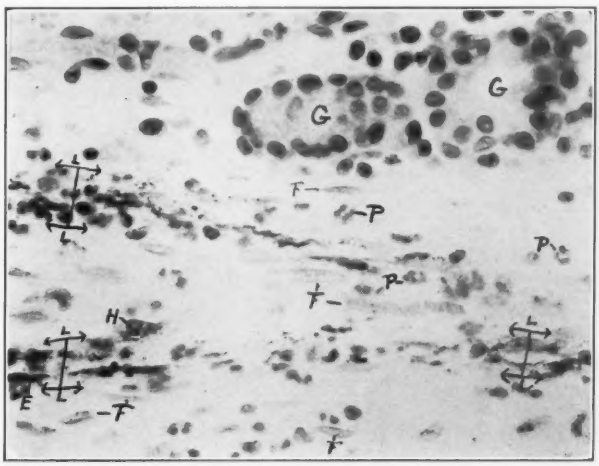

Fig. 6. Subepithelial tissue showing black carbon striations along the capillary lymphatics (L) and within the endothelium of the capillary walls (E). Carbon particles not taken up by mucus glands (G), fibroblasts (F) or polymorphonuclear leucocytes (P). Few granules in histiocyte (H). Hematoxylin and eosin stain. (High power magnification.)

traveling in straight lines both within and without the lymphatics, indicating the direction of spread to be from before backward, and not upward or downward, as no carbon was found in the uninjected turbinates or in the antral mucosa.

The cytologic reaction to the inert carbon particles was most interesting. The most rabid phagocytes were the histiocytes and fibroblasts, the monocytes to a lesser extent and the polymorpho-

nuclear leucocytes apparently not at all. The relation of the carbon particles to the histiocyte varied, occurring both within the cell and attached to it. (Fig. 9.) Within the cell protoplasm they were spherical, quite coarse as a rule, and of irregular size, a characteristic of pigment within phagocytic cells, and assumed a perinuclear arrangement. The nucleus did not appear to take up the carbon.

The carbon inclusions of the fibroblasts, whether wandering or fixed, were in the forms of smaller granules in the protoplasm,

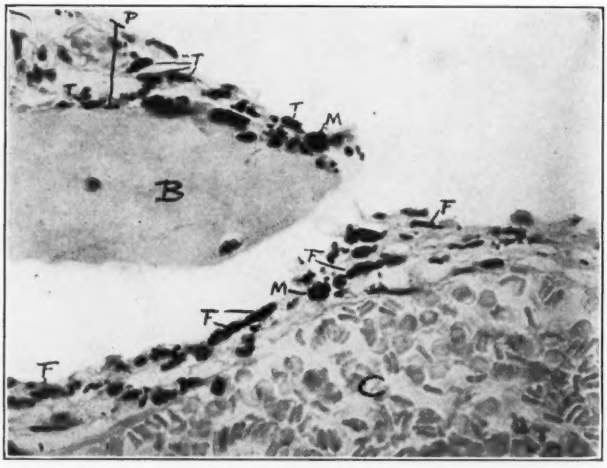

Fig. 7. Showing marked phagocytosis of carbon particles by the fibroblasts (F) and monocytes (M) about a blood capillary (C), and by the fixed connective tissue cells (T) of the periosteum (P). (B) Bone. Hematoxylin and eosin stain. (High power magnification.)

though in some mature cells the carbon seemed to be within or upon the nucleus. (Fig. 7.)

The carbon particles did not penetrate the epithelium in an effort to migrate to the surface, nor were they found within the epithelial cells, though frequently in close contact with the basal cells of the epithelium.

Rabbit 4.—These tissues showed a marked clearing of the spaces and a more pronounced accumulation of carbon particles

within the smaller lymphatics, the histiocytes, the monocytes and the fixed connective tissue cells. (Fig. 7.) Again there was no phagocytosis by the polymorphonuclear leucocytes or the epithelial cells or penetration of the epithelium by the carbon particles.

Rabbit 5.—There was an almost total absence of carbon particles from the tissue spaces. Many of the large lymph spaces were packed with phagocytic cells filled with carbon particles (Fig. 8), and the smaller lymphatics of the subepithelial and

Fig. 8. Large lymph space (L) immediately underneath the partially fragmented columnar epithelium (E). Space is filled with histiocytes and monocytes loaded with carbon. (H) Histiocytes. (F) Fibroblasts. (M) Monocytes. (E) Endothelium of lymph space. Hematoxylin and eosin stain. (High power magnification.)

deeper tissues contained more carbon, as did the fibroblasts and histiocytes than the six-hour specimens, but about the same amount as the twenty-four hour uninfected specimens. While there appeared to be an increase in the number of polymorphonuclear leucocytes in the tissues, none were seen to be phagocytosing the carbon particles. There was no tendency for the particles to migrate through the intact epithelium or to be phagocytosed by the epithelial cells.

Rabbit 6.—The microscopic picture was essentially the same as in the normal and the infected twenty-four hour rabbits, except that the lymph capillaries appeared to contain more carbon and the histiocytes were slightly more numerous and more contained carbon particles. (Fig. 9.) There were no carbon particles within the polymorphonuclear leucocytes, which were not increased in number. The apparent increase in the number of actively phagocytosing histiocytes was not sufficiently marked to lead us to believe that it was the result of the bacteriophage, either by rendering the carbon particles more assimilable or by making the cell membrane more fragile and thus more permeable.

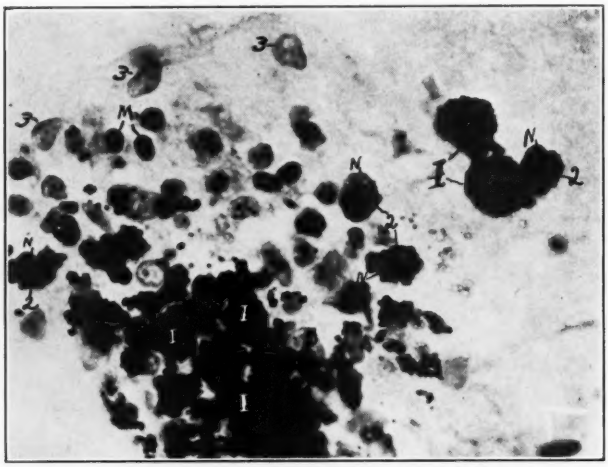

Fig. 9. Histiocytes (1) completely filled with carbon particles; (2) showing perinuclear accumulations; (3) entirely free of carbon with cell outlines not discernible, nucleus alone staining. (N) Nucleus. (M) Monocytes. Hematoxylin and eosin stain. (Oil immersion.)

It was interesting to note the presence of carbon particles within the spaces of a lymph follicle immediately posterior to the left inferior turbinate. The particles were seen free in the lymph spaces in the heart of one nodule and between the cells about the large blood vessels in an adjacent nodule. There was definite phagocytosis by a few histiocytes and monocytes but apparently

none by lymphocytes. This illustrates very clearly that foreign particles are carried from the nasal mucosa without being phagocytosed, and filtered out and phagocytosed in the first lymph nodule posteriorly.

RECAPITULATION.

It thus appears from the foregoing findings that carbon particles do not penetrate into the subepithelial spaces of the mucous membrane of the turbinates, through an intact epithelium or through an intact or a broken epithelium of the maxillary antrums, and that the ciliated columnar epithelium of these areas, whether attached or free, does not phagocytose the carbon. It is also evident that carbon particles within the antral cavities are phagocytosed by the free monocytes, young connective tissue cells and histiocytes and not by the polymorphonuclear leucocytes.

At some areas of broken epithelium in the antral mucosa there appeared to be a marked migration of fibroblasts and small histiocytes from the subepithelial tissues, but the preponderance of polymorphonuclear leucocytes in the antral cavity, with no evidence of passage through the epithelium at six hours, indicated that these preceded the histiocytes and fibroblasts to the area of irritation, as there were very few of the latter in the mass of free cells.

The lack of penetrability by inert matter and the absence of any attraction for it by the epithelium in toto or by its cells individually, would imply the necessity for an invading body to be biologically, electrically or osmotically active in order to create a condition of mutual attraction with the epithelial cells of the nasal or antral mucosa.

Our findings failed to corroborate those of Lenart, as the mucous glands, ducts and cells were singularly free of carbon particles in all of the specimens examined. (Fig. 6.)

This disparity of reactions of the epithelium of the mucous membrane of the nose and maxillary antrums, compared to the reactions of that of the trachea and bronchi and of the tonsil, strongly suggests a specificity in the defense mechanism of these respective mucosas.

The activity of the histiocytes and fibroblasts and, to a lesser extent, the monocytes in ingesting the carbon particles, as con-

trasted with the inactivity of the polymorphonuclear leucocytes in this regard, is noteworthy. The fact that the latter do not take up inert, biologically inactive matter, but do ingest living bacteria, points to a sequence of phagocytic properties in the two types of cells. The living bacteria being ingested and killed by the polymorphonuclear leucocytes, are freed by their dissolution, and both in turn are phagocytosed by the histiocyte, the fibroblast or the monocyte, which invade the site or irritation in larger numbers after the polymorphonuclear leucocytes. In the antral exudate the polymorphonuclear leucocytes appeared to be viable and capable of phagocytosis of living organisms, consequently a barrier to invasion by micro-organisms which it would be illogical to remove by premature or too often repeated lavage.

The distribution of histiocytes in the normal nasal and sinus mucosa was quite general throughout the stroma, in the subepithelial and deep tissues as well as about the lymph capillaries and blood spaces. Their phagocytic properties were but slightly increased by infection, and apparently not any more so by the bacteriophage. They occurred in the larger lymphatics, singly and in clumps, containing ingested carbon particles. (Fig. 8.) This corroborates the observations of Menkin,[22 23] that there are many occluded lymphatics at the site of inflammation and that solid particles can disseminate only with difficulty from the inflamed area through the regional lymphatics. However, this occlusion of the lymphatics was present only in the tweny-four hour specimens after the histiocytes had become active and a great deal of dissemination could have taken place prior to that time, as Menkin[24] states that the capillaries become much more permeable to particulate matter with inflammation. Histiocytes are known to be motile by reason of their pseudopods, and in all probability change their position, not only in the tissue spaces, but also in the body itself, by insinuating themselves into the lymphatics and blood capillaries between the endothelial cells of their walls, being carried elsewhere by the lymph or blood streams, respectively, probably carrying within them viable bacteria.

The fibroblasts appeared to be a more important factor in phagocytosis than the histiocytes, on account of their wider distribution and larger numbers. Carbon particles were found in the

protoplasm, not only in the apparently less fixed types of cells but also in the definite structural cells, such as those of the stroma and the periosteum, especially after twenty-four hours. (Fig. 7.) One cannot say exactly what ultimately happens to these fibroblasts. Since the carbon particles are indestructible they must survive and be freed by the breaking up of the cell, to be rephagocytosed and fixed or ultimately carried away by the lymph stream, though a connective tissue cell containing carbon can live indefinitely. The endothelial cells of the walls of the blood capillaries also contained carbon particles in some instances. (Fig. 5.)

The primary route of spread of the carbon particles occurred in the tissue spaces by dissemination. Inasmuch as the carbon particles were nonmotile, their spread could only be ascribed to the movements of the tissue fluids, which coursed in straight hair-like streams, and apparently from anterior to posterior, in turbinates which had been injected in their anterior extremities only. The carbon particles were not eliminated to the surface and through the epithelium, as in the tonsils.[10] These lymph streams all seemed to flow towards the perivascular lymphatics, but there the carbon remained within its channels and did not penetrate the blood capillaries or walls of the larger vessels. (Figs. 4 and 5.)

Whereas, after six hours there was a widespread distribution of carbon in the tissue spaces (Fig. 4) and relatively smaller amounts in the lymph capillaries (Fig. 6) and phagocytic cells, after twenty-four hours the picture was reversed, with practically no carbon in the tissue spaces and larger amounts in the lymph capillaries and phagocytic cells (Fig. 7) and in a lymph nodule posteriorly.

No carbon was found in uninjected turbinates on either side, indicating that there is probably no cross distribution or superior or inferior spread from turbinate to turbinate, and not the intimate connection between the lymphatics of either side of the nose as there is between the tonsils. Lenart[10] demonstrated this connection by injecting carbon into one tonsil and finding it in the other.

There was no information gained by the use of the bacteriophage, except that it does not stimulate the phagocytic cells to

any appreciable increase in numbers or increase in activity, whatever its alleged beneficial effects may be.

As a corollary, we may apply these observations to human beings by visualizing the subepithelial tissue of the entire respiratory tract as one continuous system from the smallest alveolus of the lung, through the paranasal sinuses, to the minutest mastoid cell, and realizing that infection may spread through these lymphatics, blood capillaries and tissue spaces, from one end of the respiratory tract to the other. Proximal spread is by direct continuity in tissue, even through bone, as this subepithelial type of tissue always accompanies the blood vessels which penetrate the bone. (Figs. 4 and 5.) Thus, for example, may develop a retrobulbar neuritis as either a localized toxic or inflammatory disturbance from infected sphenoid or posterior ethmoid sinuses, which when operated upon and opened allow free drainage of the lymphatics leading to the optic nerve area. In a similar manner, by extension along the subepithelial tissue of the eustachian tube and middle ear, may develop a mastoiditis without a frank otitis media, especially on the same side as an infected maxillary sinus, or a meningitis may follow without an erosion of the overlying bone.

Another route of proximal spread of infection is indicated by the blockage of the lymphatics seen about the area of irritation (Fig. 8), causing a backing up and reversal of a lymphatic stream, thus carrying the invading organism either free or within the phagocytic cells farther back into the neighboring tissues. From this we can more easily understand why infection does not necessarily spread in the direction of the flow within the lymphatics or blood capillaries. The more distal spread of infection is brought about by being carried either free in the stream or within the phagocytic cells, through the blood capillaries and non-obstructed lymphatics of the perivascular zones, to a wider distribution throughout the body.

The problem of the specific phagocytic properties of certain cells in the mucosa of the nose and paranasal sinuses is not of mere academic interest. It concerns all of us as clinicians to familiarize ourselves with the phagocytic phases of resistance to infection, if we are to think and act intelligently in combating infection. There is an optimum time to irrigate an antrum or

to operate upon an infected sinus, and the stage of phagocytic activity of the various specific cells should be an important determining factor. Of the manner of stimulating the formation and activity of phagocytic cells, our knowledge is extremely limited.

It is obvious, therefore, that it is necessary to have other elements present besides the physical contact of foreign matter with the turbinal or sinus epithelium in order to incite invasion of, or phagocytosis by, the epithelium and the polymorphonuclear leucocytes. Whether the necessary complement is osmotic tension or biochemical or electrical influences, and what proportion of each is essential, are problems for further investigation.

SUMMARY.

1. Carbon particles in suspension do not penetrate the intact ciliated or squamous epithelium of the turbinates of the rabbit after thirty minutes to six hours' contact.

2. Carbon particles in suspension do not penetrate the intact or broken epithelium of the antrum of the rabbit after six hours.

3. Neither the ciliated epithelial nor the squamous cells of the turbinates are phagocytic for carbon particles.

4. Neither the attached nor free ciliated epithelial cells of the antrums are phagocytic for carbon particles.

5. Particulate matter spreads through the subepithelial tissues of the mucosa of the turbinates and antrums in four ways: (1) Through the tissue spaces; (2) through the capillary lymphatics of the stroma; (3) through the perivascular lymphatics; (4) through the main lymphatics.

6. The direction of spread of particulate matter in the turbinates is from anterior to posterior, and not from turbinate to turbinate, or from the turbinates or antrums of one side of the nose to those of the other.

7. Particulate matter is phagocytosed in the subepithelial tissue of rabbits by the histiocytes, the fibroblasts and fixed connective tissue cells, the monocytes and the vascular endothelium. Phagocytosis is more complete after twenty-four hours than after six hours.

8. Phagocytosis of particulate matter by fibroblasts, histiocytes or monocytes is not activated by infection or by a bacteriophage.

9. Polymorphonuclear leucocytes, whether free or in the subepithelial tissue, do not phagocytose particulate matter.

634 N. GRAND BLVD.

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LI.

TACTILE SENSATION AS RELATED TO HEARING TESTING AND HEARING IMPRESSIONS THROUGH NERVES OTHER THAN THE EIGHTH.

DOUGLAS MACFARLAN, M. D.,

PHILADELPHIA.

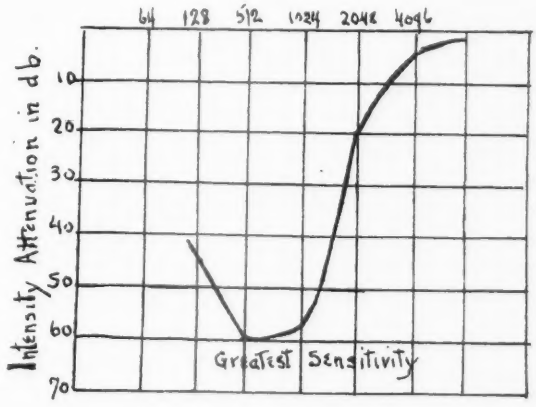
This study has been undertaken to supplement work on the methods of testing the hearing of deafened children. These children usually have some residual hearing. Many of them have become deafened before acquiring language; many, on account of severe deafness, have either poor concept of sound or have developed marked auditory inattention. In testing these children for hearing it is often quite difficult to determine whether they are reporting hearing or feeling. Most of the hearing test methods exhibit vibrations that can be felt as well as heard when used in the large intensities with which the deafened have to be tested.

Fortunately, for differentiation, the sense of hearing is more delicate than the tactile sense; and fortunately, the latter sense is relatively poor about the ear. Vibrations whose intensities have diminished below the tactile threshold can still act as strong auditory stimuli; this is particularly true of frequencies above 500 cycles.

Method Used.—The source of sound and of vibration was a beat-tone oscillator, which delivered any cycle between 16 and 8,000. The vibrations were varied both as to frequency and as to intensity. The selected cycle was passed through tubes of amplification, and then through a sensation-unit rheostat which was graded in 2 S. U. steps. The vibration was ultimately delivered to a "bone conduction receiver." This is an electromagnetic unit with a short rod applicator fastened to the armature. Where the vibrating unit was used to test the sensation on the palm or surface of the distal end of the right index finger, the subject was allowed to touch the top of the rod with varying pressures to

determine whether or not at any one time he could feel the vibration. Other points of application are noted in the records illustrated. The low figures denote the greater intensity of stimulus.

Norms of Persons with Normal Hearing.—(The sensation of the finger tip was alone used. The tactile sensation about the ear cannot be taken in normal hearing, as hearing masks the touch sense.)



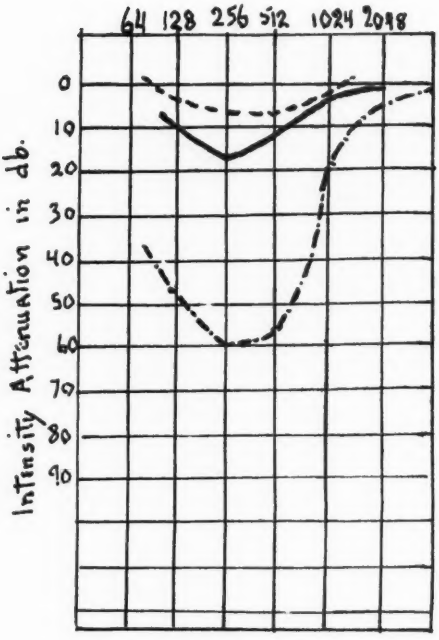
Normal tactile sensitivity from finger tips.

Tactile sensation in the deaf, taken to determine the tactile sensation about the ear.

Case 1.—Jacob W. B., age 55, deaf-blind since infancy, has a brother who is also deaf-blind. History of consanguinity; deafness and insanity in the family. No residual hearing.

Significance: A deaf-blind case, which might be expected to exhibit a cultivated tactile sense, shows subnormal tactile sense of finger tips and tactile sense of lobule.

Case 2.—Hans N., aged 44, suffered from severe Ménière's syndrome. Hearing was practically lost on the affected side (right). The left ear is relatively normal. The right eighth nerve was cut by cerebellar approach in 1928. At this time the seventh nerve was in some way completely paralyzed. In 1929, Dr. Coleman of Richmond did a very satisfactory faciohypoglossal



J. W. B. Born deaf-blind.
Tactile sense from lobule — — — —
from finger tip ————
Normal tactile sense from finger tip —.—.—.—

anastomosis. I saw the case subsequently, in 1930, and made the following tactile and hearing tests:

Hearing and Tactile Sense.—The hearing in the left ear was practically normal to single pitches and to phonographic speech. The right ear was totally deaf.

A composite sound with 256 predominating, gave tactile sensation in the left ear at 100. The audiometer used is so made as to be graded with the weakest intensity barely audible to the normal; the greatest intensity gives initial tactile sense at the ear in the hearing individuals. On the right (or deaf ear) tactile response appears at a point 15 S. U. less than at the good ear. The explanation is that loudly heard sounds mask, to that degree.

the initial perceptions of tactile sense of the normal hearing ear. Tactile responses elsewhere appeared normal.

By completely masking the bone conduction hearing of the good ear, to various pitches, by means of just sufficient noise, it was possible to prove that there was no nerve pick-up in the destroyed ear, when loud sound was delivered in different pitches to this ear by bone conduction. Two exceptions may be taken to this proof: (1) The contralateral effect of bone conduction sound occasionally heard best in the opposite ear, and (2) the central overshadowing of small response in the deaf ear due to the loud sound in the good ear filling the consciousness.

Touch Discrimination.—

Tongue and nose	2.2	Back of hand	12.0
Lips	2.5	Back of leg and shoulder	16.0
Fingertip and forehead	3.0	Abdomen	26.0
Back of finger	5.0	Sole	28.0
Palm of hand, arm, thigh	7.0	Back of forearm	33.0
Forearm	8.0	Gluteal region	48.0

Vibration Discrimination.—(Case with eighth nerve severed.)

External auditory meatus	6.6	Cheek	6.8
Pinna	7.3	Malar bone	7.5
Palm	7.0		

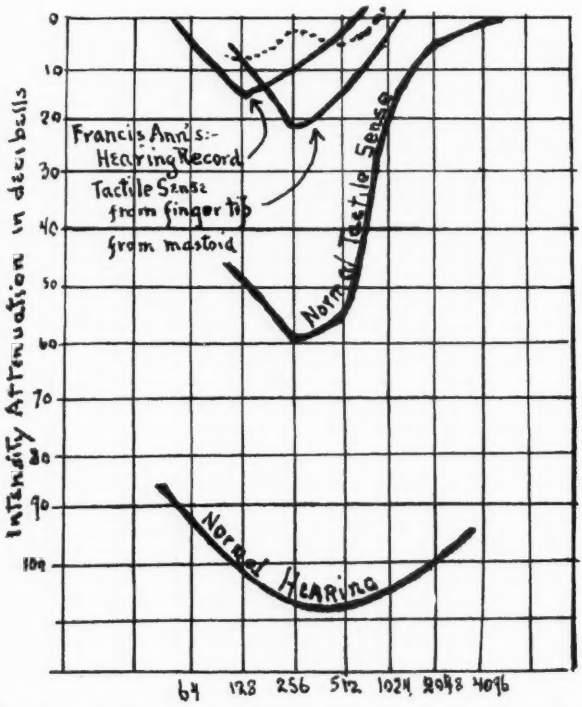
Case 3.—Frances A. D., age 15. This child has been deaf since infancy. There is a history of family deafness. She is alert and intelligent, understands written language well, and seh can read lips with fair ability. Her speech is difficult to understand, and is altered because she neither hears her own voice nor that of others. However, greatly amplified speech sounds she can hear and understands without lip reading. This fact is true in spite of common knowledge that marked deafness produces such auditory inattention that auditory word images and memories are apt never to form.

Her ears, nose and throat are negative. The tubes are open.

At the outset of testing, I have determined that she fully understands the purpose of the tests—that is, that I wish to test her hearing. She proves by initial testings that she can distinguish between hearing and feeling. She can recognize by her ears the difference between single tones, speech and music. Thus, without

doubt, she has concepts of sound. This is proved by the fact that she can imitate sounds presented to her in loud intensity.

She answers with unusual accuracy, even at threshold intensities; her thresholds are narrow and sharp. Malingering test methods are frequently interposed to insure accuracy and reliability in reporting.



Tactile sense and hearing in Frances Ann D. (Congenitally deaf.)

HEARING.

Beat Tone Audiometer.—Both ears show approximately equal loss; neither hears the 64 cycle nor the 2048 cycle. The loss for pitches 128, 256, 512 and 1024 runs evenly between 84 and 98 S. U. loss—profound deafness.

A beat tone oscillator is used to deliver any desired pitch amplified to her ear. With this receiver at her ear and with forks touching her teeth she can exactly match, by hearing, pitches 128, 256, 512. She is correctly conscious of small pitch variations.

Fork Hearing.—Nothing by A. C. in either ear with any of the forks. B. C. markedly reduced in both ears; can hear nothing by B. C. above 512.

Speech Hearing.—Right ear has a sharp threshold to amplified speech at considerable intensity, 80 S. U. louder than normal threshold. The left ear also has a sharp threshold to speech at 64 S. U. louder than normal threshold.

Touch Hearing.—This girl exhibits the singular phenomenon of being able to perceive sound as sound through touch. With her finger tips on the reed of a loud speaker unit she can recognize separately phonographic speech, instrumental music, singing or single tones. To the latter she can exactly match single tones delivered to her ear. She can discriminate by her fingers, a raising or lowering of single pitches. Her intensity discrimination from the finger tips is poor as is her general tactile sensibility. Recall that she is deaf to sounds except those of the loudest intensity; she cannot hear by her ears the faint sound air borne from the unit.

That she does not receive the sound by bone conduction is evident when there is no perception if the unit is placed against the olecranon. Yet there is perception a fraction of an inch away from the ulnar nerve in its groove.

With the fork handle on the finger tips she discriminates between the vibration and sound she perceives. The latter fades first. The fact that she reported both hearing and feeling the fork, led me to discover this phenomenon.

The only explanation that can be ventured is that there remains, in the crude tactile, that primitive auditory capacity which through evolution finally became the special sense organ of hearing. We should, however, expect no central sound imagery to be developed from this source; the appearance of this imagery would suggest that the stimuli go to centers already trained by true audition.

In frequent examinations of the deaf, there has been occasionally suggested that the patients had this tactile hearing. But I have never before been sure that a true concept of sound was present. Deaf patients are prone to report feeling for hearing. A convenient discriminating test is one described by the author.[3]

A sounding 512 tuning fork is held erect with flat of the tine facing the pinna, the edge is toward the external auditory canal. The child is trained to hold up his hand as long as he notices anything at his ear. Vision, of course, is eliminated. The strongest auditory and tactile impulses come off the flat sides of the fork. When the hand is dropped, the flat of the tine is rotated to face the meatus. If the hand is again raised, the child is reporting hearing since the increment of tactile sense is negligible but the increment sound is great.

Gault's work in 1926 and subsequently[2] does not cover this phenomenon. He has shown the possibility of training the pure tactile sense to distinguish words which are setting up vibrations on a diaphragm. Tactile-word-memories are built up.

Dr. Shambaugh's[4] case of hearing without a cochlea stands out as unique. The observations and testings were carefully made, and there is reason to believe the conclusions. One of the cochleae in this case was discharged in a sequestrum. The good ear was dampened out, and there was sound pick-up from the damaged side. The suggestion is that the eighth nerve itself picked up the stimulus, though recent experiments with the Wever-Brey technic contradict this possibility.

The phenomena described by Eichhorn and Meyer of hearing from electric terminals without the telephone receiver is fully explained on physical grounds, and need be mentioned here only.

Knudsen's[1] work on the tactile appreciation of vibrations published in 1928, is most thorough and complete. He has been able to get responses to vibrations of 2.5 dv as the lower limit. The lowest frequencies that have been detected by my experiments have been around 64 dv. His upper limit was approximately 1600 dv, with the suggestion that it "is certainly higher than this, possibly as high as 3000 dv to 4000 dv." I have been able to get response at 2400 dv. Perception at both upper and lower limits

depends greatly upon the intensity of the stimulus, just as it does with hearing. Both Knudsen and myself have found the maximum response to be around the frequency 256.

Intense vibrations, 2,500 times the strength of minimum threshold amplifications (at 64 dv), produce extreme fatigue and after-pain in the finger.[1] Thus there is a pain zone analogous to the hearing pain zone at great sound intensities.

Discrimination of differences in amplitude for various pitches "is approximately the same for various pitches, and the smallest discernible change in amplitude decreases as the amplitude increases, approaching a constant value of about 5 per cent."[1] In other words, as one gets in the region of easy tactile observation, closer amplitude discrimination appears.

As to frequency-difference-discrimination, the changes must be gross before appreciated (15 to 30 per cent).[1] The ear can, however, detect infinitesimal changes. With touch "probably as many as twenty or thirty gradations of frequency can be distinguished between the limits, 16 dv—1600 dv., whereas the ear can sense 1000 changes between these same limits."[1]

With the tactile sense as with vision and hearing, single stimuli have to be presented at a greater rapidity than 16 times per second in order to appear as a continuous emission. Thus the "flicker" sense is identical for all.

The following observations are recorded after the examination of many subjects who are both partially deaf, severely or totally deaf, or who have normal hearing.

1. The deaf have no greater tactile sensation than the normal have, yet, in occasional cases, their perceptive sphere is much more acute as is shown by the sharpness, narrowness and accuracy of the threshold. They are often for very good reason, much more "touch-conscious" than the normal individual.

Illustrative Case (Not Tested Quantitatively).—A young boy, age 7, was struck by an automobile and suffered severe cranial injuries that totally destroyed both sight and hearing. When seen three months after the injury, he was going about feeling and smelling everything. He thus was developing new memory-knowledge of things through the sense of touch and smell. (See:

Studies in Tactile Sensation in the Blind, C. F. Ferree, Amer. J. of Psych., 1906, XVII, 81. XIX, 59, 484.)

2. The amount of pressure of the vibrating body upon the skin introduces a considerable factor of variation. With the finger, the subject was allowed to use varying pressures at will, so as to select the optimum pressure. Considerable difficulties present themselves in inventing any method of accurately standardizing or controlling pressures. The armature of the vibrating unit may be considerably dampened by heavy pressure.

3. Considerable variation occurs in the testings of various individuals, so that there is merely a "wide normal." These differences are inherent variations without obvious cause or explanation.

4. After-images of tactile sensation are more conspicuous and more common than auditory after-images. In some cases, the vibration image or memory is so persistent as to make any testing accuracy impossible.

5. Association Concepts.—A peculiar mental concept or translation of the tactile sense is frequently noted in certain individuals of special training or experience, such as musicians, experimenters in acoustics, psychologists. The tactile vibration may set up a vague tone image. This image is never identified by them as a true tone capable of imitation. Yet so frequently with such people is this vague concept produced that the occurrence is noteworthy.

6. Variations in tactile vibration intensity can obviously be detected within the limits. The nearer the minimum threshold, the less keen is the intensity-difference-discrimination. Normal individuals vary greatly, not only as to threshold, but as to intensity-difference-discrimination.

7. Frequency discrimination by tactile sensation does not occur unless the stimuli are "crude," strong and widely separated. Training may considerably exalt the capacity; associated memories will also cultivate the faculty.[2]

8. Response to Various Frequencies.—As with hearing and vision there is a macula of greatest perception. Certain frequencies are felt best. Above and below this macula (as with hear-

ing) there are indefinite zones of weaker perceptions—that is, frequencies somewhat above and below those best felt may be perceived if their intensity is made sufficiently great.

9. The absolute measure of tactile-threshold-intensity of frequency 256 (center of macula) can be expressed as 50 S. U. above the auditory threshold for this pitch. (The Sensation Unit is the most available and comprehensive measure at hand. Further, it happens, fortunately, that the frequencies most easily felt are in the zone of those most easily heard.

10. It is essential in testing the deaf that we keep in mind the possibility that the patient's response is not to hearing but to feeling. Methods must be used to make certain that there is no confusion.

1805 Chestnut St.

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LII.

OTOSCLEROSIS IN A SEVEN MONTHS FETUS.*

Louis K. Guggenheim, M. D.,

St. Louis.

The earliest cases of histologic otosclerosis heretofore reported[1] are two at 5 years (Siebenmann, O. Mayer), 3½ years (Manasse, 1915), and 1 year, 10 days (Guild, 1930). The present case, to be reported fully at a later date, and here published as a matter of record, is that of a seven months' fetus. The lower right photomicrograph is of a normal area in the enchondral capsule. The three other views show various parts of the otosclerotic focus. The condition being at an earlier stage than ever before encountered, presents unusual histologic details. The dark areas are all typical blue staining otosclerotic fiber bone.

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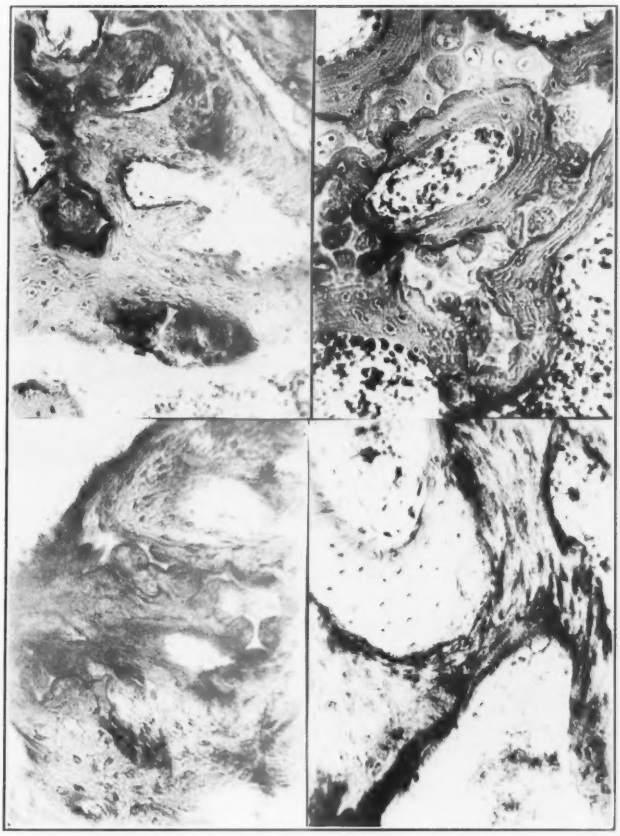
*From the Oscar Johnson Institute of Washington University.

1. Guild, Stacy R.: "Early Stages of Otosclerosis." Archives of Otol., Oct., 1930, Vol. 12, p. 25.

Note: The next article of Dr. Guggenheim's series on "The Cause of Otosclerosis," will appear in the December Annals.—Ed.



LIII.

CONGENITAL ATRESIA OF THE EXTERNAL AUDITORY MEATUS.*

LYMAN RICHARDS, M. D.

BOSTON.

The condition of congenital atresia of the external auditory canal has been an object of interest and investigation to three distinct groups of medical workers. Anatomists and embryologists have made elaborate and searching studies into the development of all portions of the auditory tract, general and particularly plastic surgeons have wrestled with the correction of external deformities so often associated with the atresia and more recently otologists have made increasing efforts to ameliorate the hearing difficulties presented in this condition. As so often happens, these groups have pursued their works independently of each other, in spite of the fact that the successful solution of the problem of any congenital defect rests on a thorough understanding of the developmental processes concerned in it. Unless the otologist, for instance, has a clear idea of the embryological processes upon which atresia of the external auditory canal rests, he will be unprepared for certain findings and situations encountered in his operative efforts to restore deficient hearing. Moreover, he will be less able to select cases in which his work will be most likely to be successful, and will be disappointed by results which may be even worse than those he sought to remedy.

Otological literature contains numerous case reports and accounts of operative procedures on congenital atresia which makes it obvious that the operator had but an imperfect idea of the developmental history of the condition and because of this was handicapped in his therapeutic attack. On the other hand most of the most comprehensive embryologic studies of the condition lay little or no stress on the relation of the development of the atresia to the subject as a clinical problem, and hence there exists a gap

*Read before the Eastern Section of the American Laryngological, Rhinological and Otological Society, Boston, Mass., Jan. 6, 1933.

which must be bridged, if the clearest possible understanding of the subject is to be obtained. Such questions as whether in congenital atresia of the external auditory canal one will always find a deformed auricle and vice versa, whether a normal drum membrane is possible under these conditions, whether or not a normal middle ear is likely to exist and particularly whether the internal ear will necessarily take part in the congenital affection are all to be found in modern otologic textbooks answered in so vague a way as to leave one uncertain as to just what are the fundamental principles on which the condition rests.

For this reason it has seemed to me desirable to compile from both the clinical and embryological works of those who have been interested in this problem a concise account of the essential developmental factors concerned in meatal atresia so far as they bear on attempts to combat the condition from the standpoint of aural surgery.

In order to do this one must consider briefly the development of the auditory apparatus as it first appears in the embryo throughout practically the entire vertebrate scale. The earliest structure to appear is the future membranous labyrinth or internal ear, which develops as an invagination of ectoderm from the surface epithelium. This soon loses its surface connection and comes to lie (Fig. 1, Vesicula auditiva) as a completely closed vesicle lined

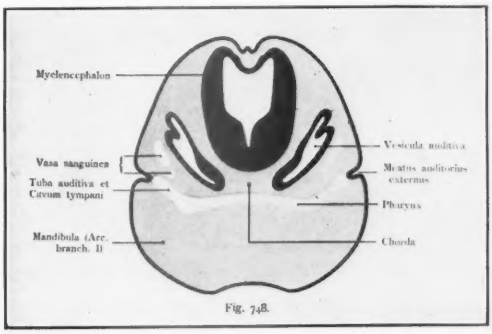


Fig. 1. Diagram to show the earliest relationship between the first pharyngeal pouch and the primitive external meatus; also an early stage of the formation of the otocyst. After Kollmann (8).

with ectoderm called the otocyst. From this develop all the complicated structures associated with the cochlea and semicircular canals. Embryologically they have no connection with any of the external or middle ear structures which all develop at a much later date. Defects in the middle or external ear are therefore independent of the inner ear, though it is quite possible for the latter to exhibit developmental defects in its own right, as in the case of the congenitally deaf child. The fact that in certain cases of meatal atresia a postmortem histologic examination has shown abnormalities in the cochlear structures is to be regarded as a coincidental finding and not as a change dependent on any intimate association in the development of these independent structures.

A consideration of the early development of the middle ear rests fundamentally on our understanding of what are known as pharyngeal pouches. These are a series of outpocketings of the entoderm lining the primitive digestive tract, of which the first or most dorsal takes part in the formation of the middle ear.

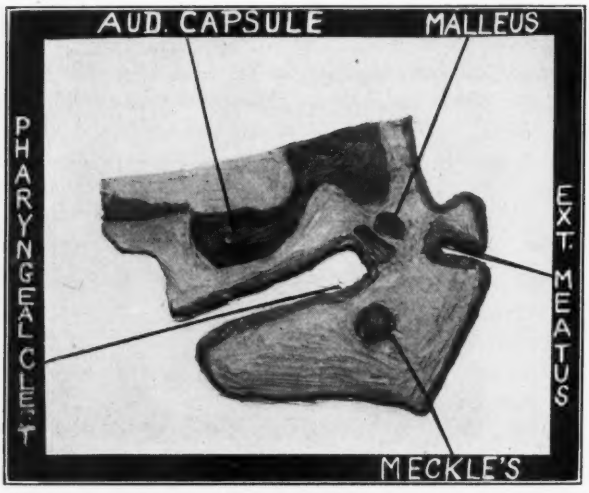


Fig. 2. A model showing the relations of the pharyngeal cleft, external meatus, with the primitive drum between them. After Kollmann (8).

This outpocketing or gill cleft, as it is known in the lower vertebrates, extends laterally into the mesodermal tissue of the head, its expanded lateral end representing the future middle ear, its internal end becoming narrowed to form the tubo-tympanic canal (Fig 1, tuba auditiva). At the same time there appears a pit-like indentation of the external skin surface in the region which will represent the future auricle. This pit extends inward for a short distance toward the first gill cleft in such a way that a narrow zone of mesodermal tissue lies between the two. This tissue, which becomes broken through as an open gill cleft in the fish is the primitive tympanic membrane, though it actually plays only a very small rôle in the formation of the future definitive drum. (Fig. 2).*

A most important development next follows and one on which to a very large extent rests the condition known as congenital atresia of the external auditory meatus. At the inner end of the

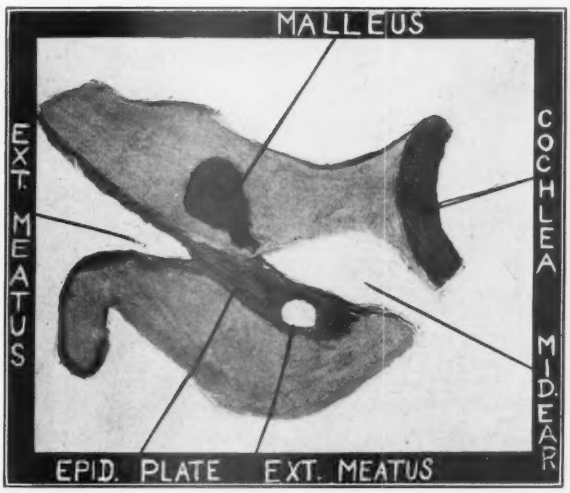


Fig. 3. A model to show the ingrowing cord of ectodermal cells to form at medial end the ectodermal disc or plate. Canalization has begun at the point marked "ext. meatus." After Kollmann (8).

*This and other models were constructed from colored plasticine by the author.

ectodermal invagination marking the first gill cleft externally, there extends inward a solid cord or plate of ectodermal cells. (Fig. 3.) This cord of cells becomes expanded medially into a disc-like plate, lying on an oblique plane just to the outer side of the lateral wall of the middle ear cavity. (Figs. 6 and 7.) The next process is the canalization from within outward of this disc, which splits into two layers. (Fig. 4.) The inner layer comes to lie against the entodermal wall of the middle ear, with a thin layer of mesodermal tissue between them, thus forming by future thinning and expansion in different directions, the definitive drum

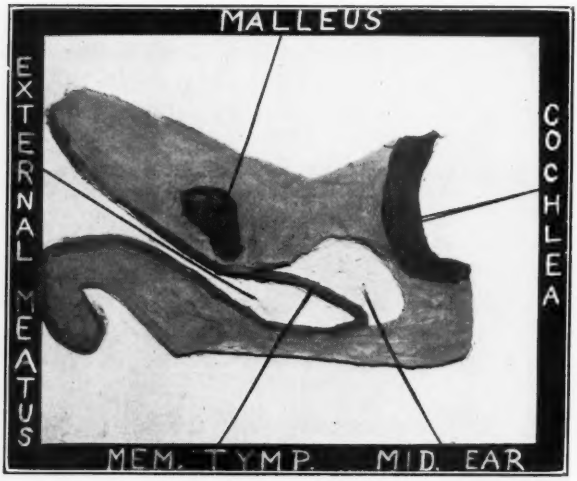


Fig. 4. Canalization of the ectodermal plate is now complete, with formation of the definitive membrana tympani, and communication between the canalized ectodermal plate and the external meatus. Modeled after Kollmann (8).

membrane. This canalization continues outward until the intervening cord of ectodermal cells becomes a hollow tube and finally breaks through to become continuous with the invaginated pit formed by the ectoderm of the external gill cleft. This process of canalization of a solid tissue has its counterpart in the development of other hollow structures in the body, notably the urinary

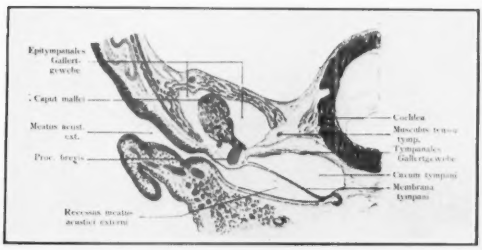


Fig. 5. The same, showing more in detail the relationships to the surrounding structures. After Kollmann (8).

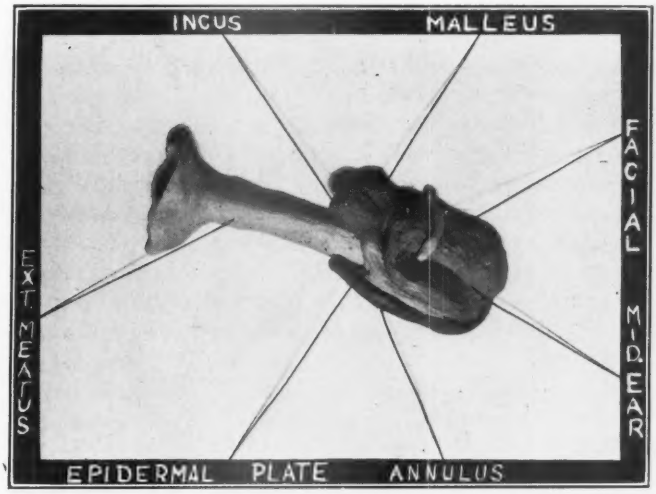


Fig. 6. Model to show the relation of the ectodermal disc or plate to the middle ear and annulus, before canalization has taken place. After Kollmann (8).

tract. Fig 5 shows this situation after the split has become complete, and the external meatus formed in the normal manner.

The development of the auricle itself is almost self-evident on perusal of Fig. 8. Around the external meatus there appears a group of cartilaginous, skin covered mounds which, in nature's inexplicable way, finally arrange themselves so as to form the concha, tragus, helix and associated structures. Further details

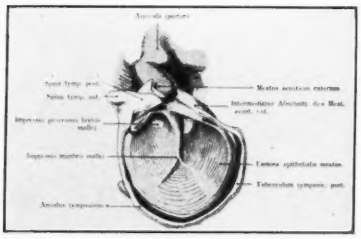


Fig. 7. The same, seen from the middle ear aspect.
After Kollmann (8).

of their arrangement and growth are beyond the scope of this paper.

We come now to a consideration of the development of certain structures intimately associated with that of the drum membrane and middle ear, the auditory ossicles. To understand the origin of these, one must appreciate the growth and development of what are known as the branchial arches. These are primitive cartilaginous paired arches situated on the ventral side of the body, laid down in mesenchyme and covered with ectoderm. In a typical lower vertebrate these arches number seven, and between them are spaces corresponding to the gill clefts. It is only the first two of these arches with which we are concerned. The first visceral

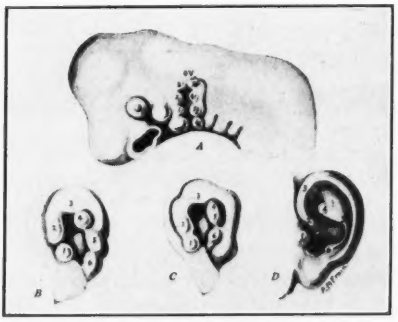


Fig. 8. Diagram to show the formation of the auricle at different stages. *A*, at 11 mm.; *B*, at 13.6 mm.; *C*, at 15 mm.; *D*, adult; *af*, Auricular fold; *ov*, Otic vesicle; *1-6*, elevations on the mandibular and hyoid arches which respectively become *1*, tragus; *2*, *3*, helix; *4*, *5*, antihelix; *6*, antitragus. After Arey (9).

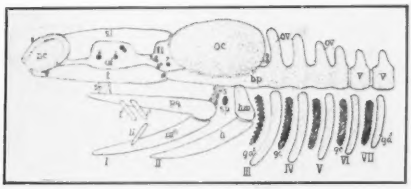


Fig. 9. Diagram of early elasmobranch skull to show the arrangement of the visceral arches. *bp*, basal plate; *c*, trabecular cornu; *fl.*, foramen lacerum; *ga*, gill arches; *gc.*, gill cleft; *h*, hyale; *hm*, hyomandibula; *L*, upper labials; *LL*, lower labials; *m*, Meckel's cartilage; *nc*, nasal capsule; *oc*, otic capsule; *of*, orbital foramen; *ov*, occipital vertebrae; *pq*, pterygo-quadrate; *s*, suspensor ligament; *sp*, spiracle; *sl*, sphenolateral; *t*, trabecula; *v*, vertebrae; *I-VII*, visceral arches; *1-10*, cranial nerves. After Kingsley (10).

or branchial arch is seen in Fig. 9 (I). It consists of a > shaped structure, the upper portion (pq) known as the quadrate, the lower (m) as Meckel's cartilage. The second or hyoid arch (II) is suspended by a small hypomandibular cartilage, itself attached to the base of the skull. It is the representatives of these cartilaginous arches in the human which, by processes of modification and developmental changes, give rise to the auditory ossicles, the malleus, incus and stapes. The changes are not always easily followed through their development in the vertebrate scale but many intermediary animals between the fish and the human show progressive steps. Thus the skull of the lizard Sphenodon in Fig. 10 show's Meckel's cartilage more nearly in its human relationship with the quadrate element fused with the skull.

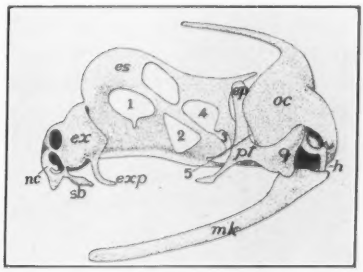


Fig. 10. Showing the articulation of Meckel's cartilage with the quadrate bone in the lizard Sphenodon. *mk*, Meckel's cartilage; *h*, hyoid; *q*, quadrate. After Kingsley (10).

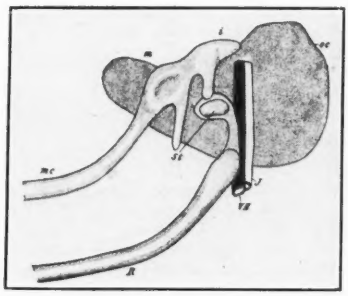


Fig. 11. Diagram to show the development of the malleus and incus from Meckel's cartilage. *i*, Incus; *j*, jugular vein; *m*, malleus; *mc*, Meckel's cartilage; *oc*, capsule of otocyst; *R*, cartilage of the second branchial arch; *st*, stapes; *VII*, facial nerve. After McMurrich (11).

In spite of certain facts to the contrary, most embryologists are agreed that the ossicles known as the malleus and incus are developed as modifications of the inner end of Meckel's cartilage. Their mode of formation is shown in Fig. 11. Some authors, in consideration of the joint normally found between the incus and the malleus, like to regard the quadrate bone, articulating as it does in lower forms with Meckel's cartilage, as the homologue of the incus, but no such relation can be established in the human. Once the malleus and incus have been split off from Meckel's cartilage, the remainder of this structure becomes incorporated in the bony structure of the mandible and practically disappears as an independent structure. (Fig. 12.)

The role played in the ossicular development by the hyoid arch is one about which there has centered considerable discussion and differences of opinions. The majority of embryologists now agree that the medial end of the hyoid arch, to which the name of Reichert's cartilage has been assigned, gives rise to the future stapes. (Fig. 13.) Serious consideration has heretofore been given the opposing claim that the stapes or at least its footplate arises as an ossification center in the cartilaginous capsule surrounding the otocyst or labyrinth, explaining to some extent the commonly noted fact that the stapes is far less likely to be in-

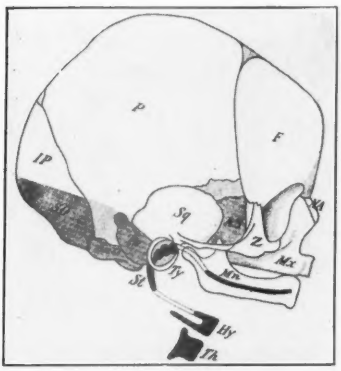


Fig. 12. Diagram to show the final fate of the first two visceral arches. The unshaded bones are membrane bones, the heavily shaded represent the chondrocranium, while the black represents the branchial arch elements. *AS*, Alisphenoid; *ExO*, exoccipital; *F*, frontal; *Hy*, hyoid; *IP*, interparietal; *Z*, zygomatic; *Mn*, mandible; *Mx*, maxilla; *Na*, nasal; *P*, parietal; *Pc*, periotic; *SO*, supraoccipital; *Sq*, squamosal; *St*, styloid process; *Th*, thyroid cartilage; *Ty*, tympanic. After McMurrich (11).

volved in the developmental anomalies of the auditory apparatus than the other two ossicles. The remainder of the hyoid arch, after development of the stapes, has a particular fate. Its proximal part, known as the latero-hyale, produces the future styloid process and becomes fused with the base of the skull anterior to the point of exit of the facial nerve. (Fig. 14.) This latero-hyale, as will be seen later, comes to play a most important part in the development of the more typical forms of congenital meatal atresia. The intermediary portion of the hyoid arch atrophies to a fibrous cord, the stylo-hyoid ligament, and the remainder becomes incorporated with a lower arch to make up a portion of the hyoid bone. (Fig. 12.) Accepting the theory of origin of all three ossicles from the first and second branchial arches, the manner in which they become incorporated within the middle ear cavity is quite clear. Lying at first entirely outside this cavity in the surrounding mesoderm, the three ossicles by a process of thinning out and expansion of the entodermal lining of the tympanic cavity, come eventually to appear to lie within it, just as the small intes-

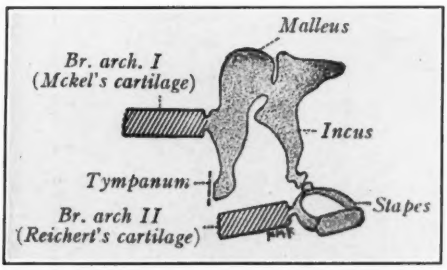


Fig. 13. Diagram showing the origin of the ossicles from the first and second visceral arches. After Arey (9).

tine appears to lie within the peritoneal cavity, though in reality it is extra-peritoneal, just as the ossicles are extra-tympanic. They are covered with entodermal membrane and in places are attached by tissue bands comparable to the peritoneal mesentery.

The os tympanum, or annulus, is the final structure which contributes to the formation of the normal drum membrane and external auditory canal. It arises as a membranous bony ring, never laid down, as are the branchial arches, in primary cartilage, and develops as a U-shaped margin about the ectodermal plate described above. (Fig. 6.) In the normal process of growth it continues to surround the drum so as to serve as its supporting attachment, but remains open superiorly in the region of Shrapnell's membrane. Subsequent external bony outgrowths from the tympanic bone contribute to the formation of the sides and floor of the bony external auditory meatus, and together with a lateral outgrowth from the tympanic roof, to that of the roof of this structure.

When all of the above described developmental processes proceed in orderly fashion and to the extent to which nature intended there is achieved the normal appearing auricle, a patent external membranous auditory canal supported in its inner portion by a bony auditory meatus, a normal drum membrane, and a set of auditory ossicles correctly located in the tympanic cavity, and functioning properly as a sound conducting mechanism to the labyrinth.

PATHOLOGY.

The fundamental disturbances in these natural processes which lead to the occurrence of congenital defects in the auditory apparatus are as much a mystery in this connection as with such other malformations as cleft palate, syndactylism or hypospadias. The wonder is that in the complicated structure of the human body they do not occur more frequently. It has been estimated[1] that congenital atresia appears in one in 2000 patients suffering from any aural disease and that one in every six of these is bilateral. A second glance at Fig. 8 will make it apparent that no severe derangement of embryonic structure is needed to produce a malformed auricle. Once any of the six basic cartilages get started on the wrong track it is quite easy to understand the final production of the familiar misshapen and deformed external ear. The variety of these abnormalities is endless, ranging from simple microtia with relatively symmetrical structure to such gross malformations as to be hardly recognizable as an ear.

The absence of a patent auditory meatus rests fundamentally on a disturbance of the above described process of canalization of the ectodermal cord which extends inward from the depression representing the external gill cleft. If this canalization fails to take place or if, having begun, it fails to extend to completion and thus to make connection with the external world by joining the gill cleft depression, then there will be a soft tissue occlusion of the canal which will be permanent. Never, so far as any reports of previous investigators go, has a normal meatus been found which is simply separated from the outside world by a thin skin-like diaphragm.

In two cases reported by Dean[2] and Colver[3] there was discovered a small sac-like structure lined with epithelium, just external to the middle ear cavity, which undoubtedly represented in each case a beginning canalization of the ectodermal cord, but it had proceeded to such a slight extent as to be quite unworthy of being designated as an external auditory canal. If now in addition to this failure of the ectodermal cord to canalize there is likewise a failure of the expanded ectodermal disc (Fig. 7, lamina epithelialis meatus) to split into its two tympanic and meatal layers

(Fig. 4), then there will be no normal tympanic membrane formed. In some instances the process of splitting may have proceeded to the point where a layer of thick, misshapen tissue occupies a position analogous to the drum, but no operator has ever reported the finding, in a case of external meatal atresia, of a normal drum membrane. Nevertheless, remarks in discussions of this subject in case reports and in otological meetings frequently give the impression that a normal drum membrane may possibly be found. Embryologically it would appear to be impossible and any operation designed to expose such a structure is doomed to failure.

Associated with the failure of this epithelial disc to split and develop a drum membrane is an abnormal development of the os tympanum. This most frequently manifests itself as a total absence of the bone or in some instances a marked hypoplasia. Under these circumstances it naturally follows that the lateral prolongations which normally spring from this bone to constitute the external bony meatus will be absent and there will be no external bony canal. Case reports vary as to the condition of the tympanic bone, but the majority describe it as either absent or greatly underdeveloped.

The condition of the middle ear cavity in the presence of meatal atresia has almost invariably been found to be abnormal in certain respects. It is apt to be narrowed in the lateral diameter and to be crossed by bands of cicatricial tissue or bony spicules. Attention naturally centers most directly on the condition of the ossicles. The malleus and incus are almost invariably present, but practically never in their normal formation. They are frequently found fused together, a fact which bears out their common origin as offshoots from Meckel's cartilage. I have found no report where there was any evidence of a persistent attachment to a residual Meckel's cartilage, though the point represented by this attachment, the processus gracilis of the malleus, is often found unduly hypertrophied. Further abnormalities in the shape and size of both incus and malleus are often found and of course the relation of the latter to what would normally represent the tympanic membrane is entirely abnormal.

The eustachian tube, originating as it does as an extension of the first pharyngeal pouch, is usually normal, anatomically in such cases as have been examined histologically, clinically in those instances in which it has been investigated from this standpoint.

The stapes shows a tendency to retain its normal appearance more often than either of the other two ossicles. The crura have been found fused together, and histological examinations have shown an abnormal incudostapedial joint.

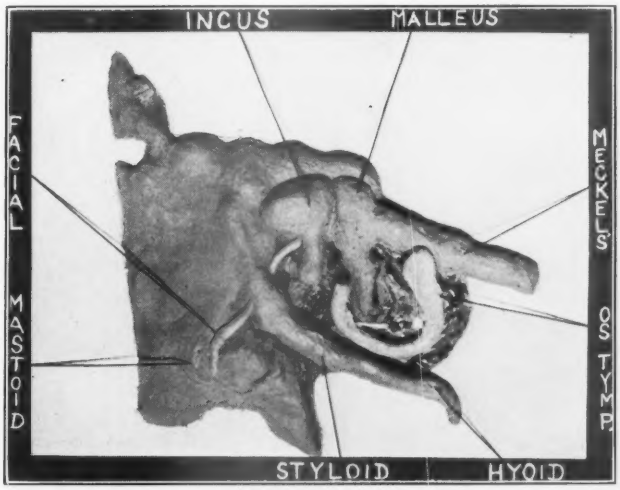


Fig. 14. Model to show the development of the ossicles, styloid and os tympanum from the first and second visceral arches. Modeled after Kollmann (8).

The microscopic changes found in the labyrinth in certain cases of congenital atresia are somewhat far afield for discussion in this paper. The opinion has been advanced that in most instances atresia was accompanied by a normal labyrinth, while in the deaf and dumb with a congenitally defective labyrinth, one never sees atresia. The degree of hearing which has so often been found present in many cases of atresia certainly indicates a clinically functioning labyrinth and the microscopic changes which have

been found at postmortem must often be more histologic than physiologic.

One of the most prominent features of congenital atresia, as seen at operation or postmortem, has been, aside from an atresia of the soft parts discussed above, a bony occluding plate, situated where one would expect to find the drum membrane, and forming the lateral wall of the middle ear. The origin of this occluding plate of bone has been the subject of two extensive monographs, one by G. Politzer,[4] and one by Alexander.[5] Both of

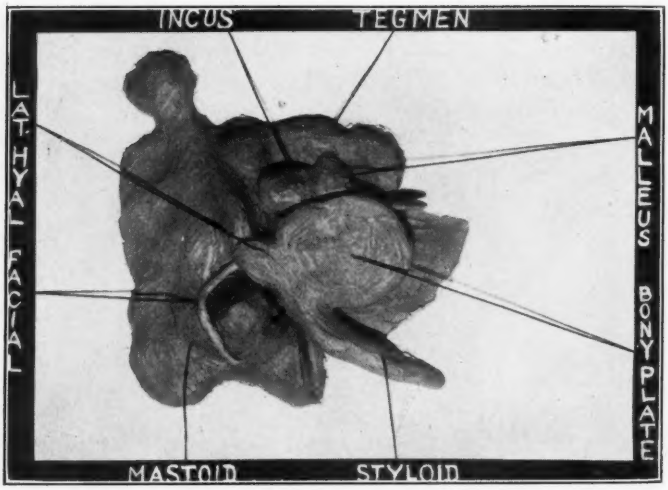


Fig. 15. Model to show the formation of the occluding bony plate from the latero-hyale. Note absence of the os tympanum. Modified after Kollmann (8).

these investigators conclude that the principal source of this occluding bone is the proximal portion of the hyoid arch to which I have referred above as the latero-hyale cartilage. From this structure, which should normally produce the styloid process, there arises a laterally extending bony outgrowth which enlarges to form an occluding mass of bone in the region of the drum membrane and annulus. At the same time this bony mass fuses with the tympanic portion of the squama and thus produces the appear-

ance seen in Fig. 16. A normal meatus is seen on the left, an occluded one on the right. It is this situation which gives rise to the clinical fact that the temporomandibular joint has been found in close proximity to the mastoid process, there being no anterior canal wall to keep it in its proper place. In Fig. 14 is a reproduction of a model of the manner in which the latero-hyale normally forms the styloid process, with no interference in the future development of the annulus or tympanic membrane. In Fig. 15 is seen the manner in which the occluding bony plate is developed

Fig. 16. Models to show the appearance of the normal external bony meatus and the same region in the presence of a congenital bony atresia.

from the latero-hyale to interfere entirely with the development of the annulus and drum membrane. This bony occluding wall is not present in every case of atresia, but is reported by a large majority of operators in accounts of their efforts to re-establish an auditory canal.

CLINICAL CONSIDERATION AND OPERATIVE INDICATIONS.

From the clinical aspect, the cases divide themselves into those in which improvement in the external auricular deformity is the main objective and into those in which the main aim is toward

improvement in the hearing function. While auricular malformation can occur, though rarely, with a normal auditory canal, and a closed canal may be found not infrequently with a normal auricle, the two are often associated together. The question of plastic repair of the auricle usually falls either to the general surgeon or to those otologists who have a subsidiary flare for plastic surgery. In either case, the patient is usually brought for treatment at a very early age, since the parents are desirous of improving the child's appearance as soon as possible. Hence the tedious task of attempted restoration is usually undertaken long before one has any idea as to the situation in regard to the hearing and before functional tests are in any degree satisfactory. Multiple operations will almost necessarily be required and even in the most skillful hands and aided by the most ingenious procedures, only moderately satisfactory cosmetic results will be obtained. Unless these cases are closely followed, and unless consideration is given to future hearing, there is danger that these patients will not receive the attention to their hearing which may in some instances prove of utmost importance.

From the standpoint of the otologist, the question of congenital atresia of the auditory canal resolves itself into the status of the patient's hearing. There is practically no other consideration which will justify surgical intervention and this is advisable only under certain circumstances. First and foremost, the functional state of the inner ear must be determined for each ear. In the presence of meatal atresia, turning tests alone will give information in regard to the static labyrinth. If, in addition, some evidence of hearing can be established, it is probable that the inner ear is functionally intact. The classical findings are those of a typical conduction deafness with raised lower limits and a Weber referred to the affected side. Hearing for voice and whisper, while reduced, will in many instances be found to be surprisingly good when the anatomical situation is taken into consideration. Some evidence as to the state of the patency of the eustachian tube should be obtained, first by examination of the tubal orifices, and second by catheterization. Several authors have established the fact that even with a closed meatus, the auscultation tube will demonstrate the entrance of air into the middle ear. If, in the

presence of a normal labyrinth and tube, the condition is bilateral and the deafness so extensive as to be a serious handicap to the individual's activities, and particularly if speech development has been interfered with, then consideration should be given to operative efforts to improve the hearing. If the condition is unilateral, then it is a mooted question as to whether any operation should be undertaken. Normal though unilateral hearing is no special handicap and will always permit normal speech development. If one were always assured of success, then unilateral operation might readily be advisable in any case. Since, however, there is always the possibility that the hearing will not be improved but even made worse, and since many of these cases have surprisingly good hearing in the presence of complete atresia, many otologists advise against any operative interference in unilateral cases. The outstanding exception to this view, as exemplified by a case of Alexander's, is the development on the side of the atresia of an acute mastoiditis, which must of course be operated upon, but by a technic which takes into consideration the congenital anomaly of an absent external meatus. Some men, notably Page,[6] having had success in improving hearing in the presence of an atresia, will naturally be more enthusiastic advocates of surgery than will be those operators whose results have not been so successful.

ROENTGENOLOGIC FINDINGS.

The use of the X-ray to determine the condition of the middle ear structure has had certain enthusiastic advocates who claim ability to detect the presence or absence of the os tympanum, the size and shape of the middle ear and antrum, and the extent of pneumatization of the mastoid process. Certain it is that many cases of meatal atresia show normal or even hyperdeveloped mastoid processes, from which finding it is certainly justifiable to conclude that the middle ear itself has not been affected in the malformation.

As might be expected, many cases of auditory atresia exhibit other congenital defects. Facial paralysis on the affected side is easily understood when one considers the proximity of the facial nerve to the structures involved in this condition. Assymetry of the face on the same side is easily attributable to some disturbance

in the bone formation around Meckel's cartilage. Other anomalies in different organs of the body have been reported in conjunction with atresia, as well as atresia in two different offspring and in a mother and child, raising the old question of an hereditary tendency to such abnormalities.

OPERATIVE PROCEDURE.

If, in the judgment of the operator, the hearing situation is such as to demand and warrant operative interference, the age at which this shall be undertaken must be decided upon. If the atresia is a bilateral one and there exists a manifest tendency to delayed speech, then it may be desirable to undertake surgical measures at an early age. Otherwise it is generally agreed that it is preferable to postpone operation until about the age of puberty, after which time there will be less tendency for any further growth of occluding tissues which might encroach on the operative field.

On at least one subject there should be complete unanimity of opinion. It is of absolutely no use to attempt any plastic reconstruction of an external meatus with the hope of exposing or restoring a drum membrane. Such an artificial meatus will invariably close and hearing will only be made worse. Moreover, any work done in the region of the normal canal line may accidentally damage the temporomandibular joint. The only possible procedure which can be of avail is a modified or classical radical mastoid operation. The mastoid must be opened and a cavity exposed down to the mastoid antrum. According to what is found at this stage, it may be advisable to attempt to remove some of the occluding bony plate lateral to the middle ear, much as one would remove the outer wall of the epitympanum in certain types of the modified radical mastoid. If the malleus and incus are so fused or deformed as to render beyond hope any possibility of effective function, they should be removed, but the stapes should be left untouched. By some form of plastic procedure on the soft parts a patent canal can be secured leading down to the antrum. Usually a skin graft will be required to assure permanent patency and even under these conditions difficulty will often be experienced in preventing cicatricial contracture and closure of

the lumen. If successful, however, sound waves will reach the inner ear through the antrum and hearing will be considerably improved in a certain number of cases. At times operators have reported a sort of drum formation over the opening into the antrum which has in some degrees served to replace the normal membrana tympani. The most conspicuous improvement in hearing following such a procedure was that reported by Dean[2] in 1920.

The most recent and comprehensive account of meatal atresia, particularly from the point of view of histological and pathological examination is that of Fraser[7] in 1931. His sections illustrate quite clearly the role played by the latero-hyale in the formation of the occluding bony plate and the changes to be found in the ossicles. There is, however, no account of the role played by the failure in canalization of the ectodermal plate in the production of the atresia of the soft parts. The present account is an attempt to correlate all these factors and to show their relationship to the clinical and surgical aspects of this interesting condition.

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LIV.

ON THE ELIMINATION OF LIPIODOL FROM THE NASAL SINUSES.

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Paris, France.

Sicard, Fabre and Forestier have recovered iodin in the urine of patients following injection of the oil into the trachea.[1] The author, in collaboration with Professor Fabre, has encountered the same finding after the introduction of lipiodol into the nasal sinuses by the Proetz method.

Experiments were conducted to study this phenomenon by means of which the possibility of the absorption of oil through other avenues could be eliminated. It was necessary to guard against the regurgitation of lipiodol into the nasal fossa, whence it could enter the pharynx, be swallowed and be absorbed by the intestine. For this purpose we employed lipiodol which had been dyed a deep blue. By this means we could watch for traces of the oil upon the upper digestive and respiratory mucosa.

In each patient, after introducing the oil by the Displacement Method, the nasal chambers and the pharynx were carefully cleansed. The oil was next shown radiographically to be confined to the sinuses, after which an esophagoscopy and a tracheobronchoscopy were done to assure ourselves that the oil had not strayed into these passages. When we had reassured ourselves that the oil had impregnated the sinuses exclusively, we collected the urine. As soon as the green color appeared, tests for iodin were begun. The blue coloring, which, as stated, was employed as a measure of caution, was found to serve a double purpose, in that its appearance in the urine invariably preceded that of the iodin. Twenty-four hour specimens of urine were examined daily for one week. The figures were remarkably constant, varying from 2.9 mg. to 3.3 mg. a day. The number of days during which this elimination occurred varied with the doses of lipiodol employed, the minimum dose in the series being 2 cc., the maximum 8 cc.

We may conclude from this that the elimination of the oil from the sinuses is not very active but quite constant. It is less than the elimination from muscle tissue, and comparable to that of the subarachnoid cavities.

The author and Doctor Bouchet, with whom these experiments were observed, are of the opinion that the Proetz method, aside from its indications in rhinologic treatment, may be of therapeutic value in general conditions requiring slow, persistent iodin therapy.

To the table of H. Roger and Binet, we may add the accessory nasal sinuses. In the decreasing order of their absorptive function, are:

The intravenous route.
The digestive route.
The tracheobronchial cavities.
Serous, pleural, peritoneal and joint cavities.
Subcutaneous cellular tissue, epidural cavity.
Subarachnoid cavities, and nasal accessory sinuses.

For the past eight months we have been studying at the Hôpital Necker and the American Hospital in Paris the variations in iodin elimination in various types of sinusitis and the relations which exist between them, on the one hand, and the elimination of iodin and the disappearance of radiopaques on the other.

We hoped to establish a supplement to the emptying test, which might give us some insight into the functioning of the mucosa. But in spite of the large number of cases which we have studied, it is not possible thus far to deduce any precise rules, as too many variables and sources of error exist, namely, comparative speed of evacuation, and the coefficient of absorption on one hand and the abnormal end results of operative procedures on the other.

Thus far we can only say that the elimination of iodin is much more brisk in the presence of an inflamed mucosa.

55 Rue de Varenne.

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LV.

OTOSCLEROSIS COMPLICATED BY OTHER LESIONS: A STUDY OF ROENTGENOGRAMS, AUDIOGRAMS, LABORATORY AND CLINICAL FINDINGS.*

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"Progressive deafness" has been quite generally thought of as synonymous with otosclerosis. In fact, all the deafness accompanying otosclerosis is commonly ascribed to bony ankylosis alone, ignoring the possibilities of other pathology in the neighboring parts. The mere fact that other lesions of the ear are not seen leads the examiner to suspect otosclerosis, and contrawise, if there has been a history of past otitis media or a definite present suppuration the examiner is prone to focus his attention upon and make his diagnoses by these lesions, consciously or subconsciously excluding the possibilities of a co-existing otosclerosis. It seemed worth while to question the accuracy of these habits of thought and to endeavor withal, by a coordinated clinical and laboratory study of all diseases of the ear, to obtain data for the study of otosclerosis itself. With these objects in view I have collected data for six years or more and now present an analysis of 126, cases diagnosed otosclerosis, all carefully studied with the aid of the latest developments in otology, including audiograms, roentgenograms and differential blood counts and chemical examinations.

Without question, some of these cases on autopsy would show no otosclerosis, and without question many cases have been omitted because of failure to diagnose otosclerosis, sign or symptom free, or sign or symptom insufficient. Also many cases have been excluded because some detail of the examination is missing or

*Presented before the Sixty-sixth Annual Meeting of the American Otological Society, Washington, D. C., May 8, 1933.

questionable. With few exceptions, my cases had been diagnosed as otosclerotics before I examined them.*

Thirty-nine cases of otosclerosis were studied by microscopic sections in complete series and also twelve in individual slides, and there is no doubt as to the lesions in these cases, but it must be kept in mind that terminal inflammations may obscure the former clinical pictures.

In all clinical studies of otosclerosis a major error threatens the accuracy of statistics, namely, the uncertainty of diagnoses. I feel that if I have erred in diagnosis it has been on the side of caution.

In this preliminary study the patients have been divided into two main groups:

1. Those without complicating nerve deafness (81).
2. Those with complicating nerve deafness (45).

In the first group only 21 per cent showed no evidence of past purulent otitis media. In the second group 33 per cent showed no evidence of past otitis media. In the whole series 25 per cent showed no clear cut clinical evidence of past inflammation in the middle ear.

Each of the two main groups is divided into those:

1. Without evidences of other complicating lesions.
2. With evidence of past masked or unrecognized inflammatory lesions in the middle ear.†
3. With definite history of past suppuration in the middle ear.

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*The pathologic research has been carried on by my son, Dr. Edmund P. Fowler, Jr., in the Department of Pathology, College of Physicians and Surgeons, and with the cooperation of the Department of Otolaryngology and supported in part by The New York League for the Hard of Hearing, Inc., and by the aid of grants from the Research Fund of the American Otological Society.

†The usual criteria were used for diagnosis, particularly positive family histories, progressive deafness, bone conduction loss near normal at lower frequencies and relatively slight compared to the air conduction loss, bilateral aspect, persistent tinnitus, pink glow upon promontory. The cases with nerve deafness, as will be seen from the audiograms, have an increased loss of hearing with the increase in frequency both by air and bone conduction, but in the lower third of the musical scale the bone conduction was within normal limits and the air conduction was distinctly down.

A fourth group logically should follow representing cases with present suppuration, as this complication is found not rarely, but I omit it at this time because these cases are not usually or readily diagnosed as otosclerosis, and they might confuse the picture.

To obtain a better visualization of the variations in the different varieties of otosclerosis all figures have been translated into percentages except under blood examination and S. U. losses, where they stand for averages.

It being manifestly impossible to go into every detail in a preliminary study, I shall limit the discussion to some of the main points in the history, the symptoms, the otoscopic examination, the roentgenograms of the head, the blood examination and the pathology shown in microscopic sections of otosclerosis at my disposal. I shall briefly point out what appears to be of interest immediately after tabulating each set of data.

Every patient suffered bilateral deafness, and in most instances one ear was affected before the other. There was no totally deaf ear. To determine this the better ear was masked by creating a noise in the external auditory canal, first simultaneously in both ears to determine the efficacy of the masking, and then by continuing the masking only in the better ear.

The first item that meets the eye in the tables is the predominance of past masked inflammations.* These constituted fully 50 per cent of the complications found in both nerve and no nerve deafness with otosclerosis. They are often unrecognized because of failure to look for the signs they leave.

With the exception of the seven children listed under past inflammation the patients were aged from 18 to 60 years. The average age was 34 in those without nerve deafness, and 36 in those with nerve deafness.

The age of onset was given as from the first to the thirty-ninth year. Omitting the children, with masked past inflammation the average age of onset was at or near 20 years in all types of complication.

*By past masked inflammation I mean otitis media which, though never recognized, has left signs of its presence, such as scars, definite retractions, adhesions, congestion or loss of movement in the membrane tensa or flaccida, etc.

ANALYSIS OF 126 CASES OF OTOSCLEROSIS IN THE THREE SUBDIVISIONS OF THE TWO MAIN CLASSES. 81 WITHOUT NERVE DEAFNESS AND 45 WITH NERVE DEAFNESS.

HISTORY		No Other Complications	No History but Signs of Past Infm. Adults	Children	Definite Hist. of Past Suppuration
Ages	No nerve deafness	18 to 40 Av. 37	21 to 40 Av. 31	1 to 20 Av. 14	18 to 51 Av. 31
	With nerve deafness	18 to 55 34	18 to 48 31		24 to 60 57
Age of onset	No nerve deafness	1 to 34 19	5 to 39 21	5 to 17 11	10 to 34 19
	With nerve deafness	16 to 25 14	8 to 37 20		1 to 22 20
Positive family history	No nerve deafness	40.6%	55.0%	36.6%	46.1%
	With nerve deafness	33.3	58.8		33.3
Progressive deafness	No nerve deafness	58.7	77.0	63.6	76.9
	With nerve deafness	78.6	50.4		66.6
Affected by colds or weather					
	No nerve deafness	18.5C 5.7W	42.5C 15.0W	36.6C 18.2W	38.5C 15.4W
	With nerve deafness	46.2C 3.2W	46.2C 8.4W		16.7C 33.3W
Exanthemata					
Measles	No nerve deafness	76.0	87.5	81.8	76.9
Scarlet Fever		12.0	12.5	50.0	15.3
Diphtheria		0.0	5.0	37.5	0.0
Measles	With nerve deafness	73.0	18.0		83.3
Scarlet Fever		20.0	5.0		50.0
Diphtheria		0.0	1.0		0.0
Colds	No nerve deafness	70.0	80.0	100.0	61.5
Pneumonia		0.0	2.5	12.5	15.3
Influenza		0.0	27.5	12.5	15.3
Colds	With nerve deafness	87.0	19.0	0.0	83.3
Influenza		0.0	0.0	0.0	65.0
A&T Op.	No nerve deafness	65.0	77.5	90.9	69.3
	With nerve deafness	80.0	84.0		83.3

The positive family histories were distinctly higher (55 to 59 per cent), in the sixty-four adults with masked past inflammation, and with the exception of this group the positive percentage was greater in those without nerve deafness. The high percentage does not necessarily indicate anything more than the inheritance of anatomic and physiologic characteristics which predispose all offspring to the inflammatory lesions suffered by their forebears.

When a cause for deafness was given it regularly appeared similar to the causes given for the types of deafness accompanying the otosclerosis. This would hardly be the case unless the first symptoms at least were caused by the complicating lesions.

A history of progressive deafness appeared in 50 to 79 per cent, varying with the complications.

The percentage of history of colds was high (62 to 80 per cent) in all types of cases without nerve deafness (and 100 per cent in children with past masked otitis), but in the nerve deafness cases was high (87 per cent) only in the uncomplicated cases and in those with a definite history of past suppuration (83 per cent), in which latter influenza also appeared to have a high incidence (65 per cent). This represents the history as given by the patients. The real percentages were probably higher, because one is apt to forget past sickness, unless it was very severe.

With all complications the incidence of diseased tonsils and adenoids with their subsequent removal was high (69 to 91 per cent). It was lowest (65 per cent) in the uncomplicated cases. This indicates a high morbidity from inflammations in the upper respiratory tract with frequent involvements of the middle ear.

Among the exanthemata measles predominated in the histories of the non-nerve deafness cases (75 to 88 per cent), and in the nerve cases (73 to 83 per cent), except under the masked past inflammations, where it averaged 18 per cent.

Scarlet fever was suffered in 50 per cent of children with masked inflammation; otherwise it was of low incidence, except with the nerve cases with past suppuration (50 per cent). Post-scarlet fever, sinusitis and otitis media often clear up with difficulty. Radiographic studies by Dr. D. S. Childs[1] demonstrate that 50 per cent of the sinuses show definite progression or extension, even after recovery from the scarlet fever.

ANALYSIS OF 126 CASES OF OTOSCLEROSIS IN THE THREE SUBDIVISIONS OF THE TWO MAIN CLASSES. 81 WITHOUT NERVE DEAFNESS AND 45 WITH NERVE DEAFNESS.

SYMPTOMS		No Other Complications		No History but Signs of Past Inflm. Adults		No History but Signs of Past Inflm. Children		Definite Hist. of Past Suppuration	
		R.	L.	R.	L.	R.	L.	R.	L.
S. U. Loss (Averages)	No nerve deafness	41.3	41.3	41.2	47.4	35.0	36.0	46.4	42.7
	With nerve deafness	71.0	74.0	61.3	63.9			65.0	65.0
Pain	No nerve deafness	24.0	18.0	37.5	40.0	27.2	27.2	38.5	38.5
	With nerve deafness	6.0	0.0	37.0	24.4			66.7	50.0
Discharge	No nerve deafness	0.0	0.0	0.0	0.0	0.0	0.0	69.3	92.3
	With nerve deafness	0.0	0.0	0.0	0.0			83.3	66.7
Tinnitus	No nerve deafness	87.5		82.5		81.8		84.6	
	With nerve deafness	73.0		67.2				0.0	
Vertigo	No nerve deafness	12.5		27.5		0.0		0.0	
	With nerve deafness	20.0		37.8				0.0	

NOTE—The figures opposite S. U. Loss indicate the average losses in each ear under the several subdivisions. The figures opposite the other symptoms indicate the frequency of these symptoms in percentages.

The incidence of diphtheria was very low or nil except in the children without nerve deafness (38 per cent). Rarely is the escape from the exanthemata complete, and seldom does the nasal pharynx and at least the tubal part of the middle ear fail to participate.

SYMPTOMS.

Deafness was perforce a constant symptom. In cases with no nerve deafness it averaged in both right and left ears slightly more than 41 S. U. loss. In the children the losses were five or six points less than this. In the ears with nerve deafness the average loss was over 70 S. U. under no complications, whereas both in the masked and definite past otitis it was over 60 S. U. The average was calculated from the losses in the areas most used for hearing speech (256 to 4082 V. D.). The audiometric losses for both air and bone conduction are shown in the composite audiograms.

Histories of pain were obtained in the non-nerve cases in about one-fourth of the ears under no complications, 38 to 40 per cent with past inflammation, and a similar percentage in those with definite past suppurations. From one-quarter to three-quarters of the patients remembered having had earache at some time, with the exception of those in the no-complication nerve group (6 per cent).

The ears with a definite history of discharge are naturally all placed under the heading of past suppuration, as perforce every patient under this division had suffered at some time a discharge in one or the other ear. In the non-nerve deafness ears there were more histories of discharge (92 per cent) in the left ear, and in the nerve ears more in the right (83 per cent) than in the opposite ears under these two divisions.

Tinnitus was present in a large percentage of ears, particularly under non-nerve deafness (from 82 to 88 per cent). In the nerve deafness ears there was no tinnitus in the cases with definite past suppuration. This may be in part due to the fact that tinnitus usually occurs in the higher tones, and in the nerve deafness cases with past suppuration, both by air and by bone conduction, the hearing for the higher tones is lowered or lost.

With the exception of deafness, tinnitus was the most constant symptom. If it occurs in both ears and there has been no other

obstructive lesion it alone suggests otosclerosis, especially if it is constant. Note that even in the children it was present in 82 per cent of cases. Tinnitus from obstructive lesions other than otosclerosis is more apt to be referred to one ear and to vary in intensity and constancy.

Vertigo in the cases without nerve deafness was present in 13 per cent with no complications, and in 28 per cent with past history of masked inflammation. In the cases with nerve deafness these subdivisions were 20 and 38 per cent, respectively. In the children and in all types with past suppuration there was no history of vertigo.

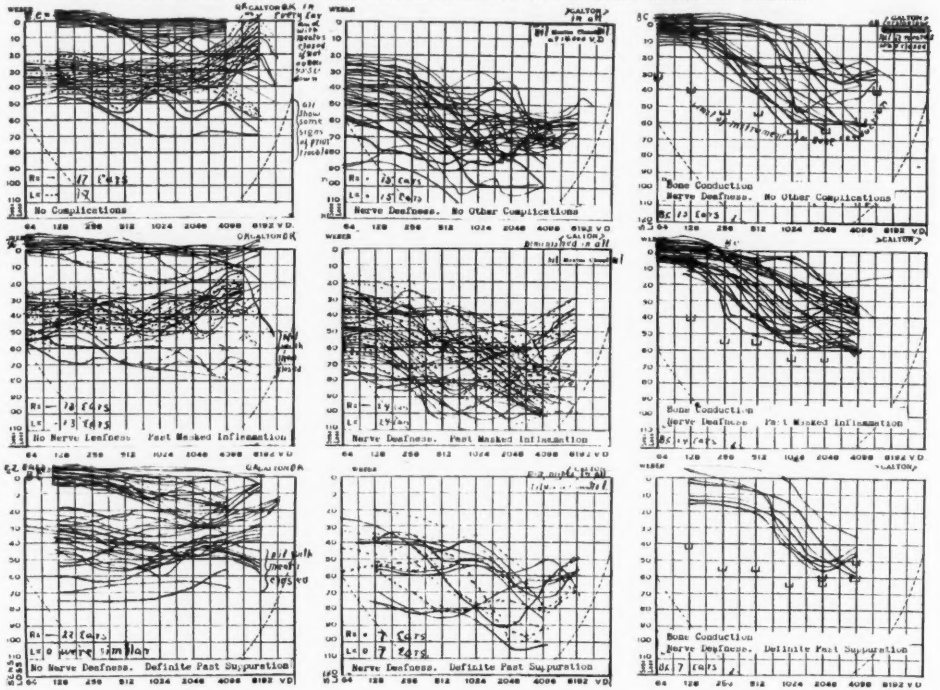
Many of my cases have been examined and tested again and again throughout many years, but in the tables but one test is given, that which was deemed most typical and exact.

The audiometric picture in otosclerosis is seen to be quite varied, depending largely upon what complications are superimposed upon the obstructive factors due to otosclerosis. Note that if the hearing for one of the tones in the lower half of the musical scale is heard below normal all of the frequencies below this are also heard below normal. I have called attention to this fact many times, but it seems that otologists persist upon testing with the very low tones, which often give false results, because it is difficult for a patient to distinguish as to whether he is feeling or hearing these tones. If either 256 V. D. or 512 V. D. is used for testing the lower tones it would be quite sufficient for arriving at a diagnosis in most instances.

It is in the upper half of the scale that the greatest variations are seen in the audiometric measurements. The hearing for the Galton whistle is indicated over each set of audiograms. The $>$ sign indicates it was heard only at a frequency below 14,000 V. D. The Galton whistle really emits simultaneously at least four different tones, and it is sometimes difficult to determine which ones are heard by the patient.

The bone conduction was near normal in every instance for the lower tones, but in the nerve deafness cases followed patterns similar to the corresponding air conduction curves. Without the bone conduction measurements at several frequencies it would

AUDIOMETRIC MEASUREMENTS OF THE HEARING IN OTOSCLEROSIS WITHOUT AND WITH NERVE DEAFNESS AND IN THE THREE SUBDIVISIONS UNDER EACH OF THESE.


No Complications
Nerve Deafness. No Other Complications
Bone Conduction
Nerve Deafness. No Other Complications
No Nerve Deafness. Past Masked Inflammation
Nerve Deafness. Past Masked Inflammation
Bone Conduction
Nerve Deafness. Past Masked Inflammation
No Nerve Deafness. Definite Past Suppuration
Nerve Deafness. Definite Past Suppuration
Bone Conduction
Nerve Deafness. Definite Past Suppuration

have been impossible in many instances to arrive at a correct diagnosis.

It is the relative losses between BC and AC which furnish the important information for diagnosis and the audiogram enables the examiner to visualize the ratio at each frequency. The Rinne test gives only a difference and not a ratio, and as I see no excuse for using the Rinne test I abandoned it long ago except for experimental purposes.

Many of the curves appear typical of nerve deafness, but in every instance the opposite ear showed no such typical picture as the higher tones were better heard, and in both ears the bone conduction was normal, for several octaves at least.

Many of these cases were tested a number of times at intervals of several months or years, and in some considerable variations in the hearing were noted. Except in those showing steady losses I feel that these variations are due more to the variations in the pathology complicating the otosclerotic ankylosis than to any change in the bony ankylosis itself. In cases showing progressive losses in hearing the increasing deafness may at times be due to extension of the obstructive or nerve lesions and not to the obstruction dependent upon otosclerotic processes.

I feel that any treatment I advised resulted in improved hearing only because it improved the pathology in the middle ear and nasal spaces. This is an approach to the therapeutic management of these cases which should not be despised.

OTOSCOPIC FINDINGS.

Congestion of the membrana tympani varied from slight redness along the malleus to intense congestion in Shrapnell's membrane. It varied greatly as between the two ears, but paradoxically was markedly higher in the nerve group of cases (67 to 83 per cent). In the children under masked past inflammation it was present in every case, and in both ears in 81 per cent.

Scars naturally were absent in ears showing no evidence of having had complications, and few in the masked past otitidies (about 12 per cent), except in children (55 per cent). In ears with definite past suppuration they were seen in 94 to 100 per cent with nerve, and 54 to 62 per cent with non-nerve deafness. These findings, of course, depended largely upon the fact that

ANALYSIS OF 126 CASES OF OTOSCLEROSIS IN THE THREE SUBDIVISIONS OF THE TWO MAIN CLASSES. 81 WITHOUT NERVE DEAFNESS AND 45 WITH NERVE DEAFNESS.

OTOSCOPY		No Other Complications R.	No Other Complications L.	No History but Signs of Past Infim. Adults R.	No History but Signs of Past Infim. Adults L.	No History but Signs of Past Infim. Children R.	No History but Signs of Past Infim. Children L.	Definite Hist. of Past Suppuration R.	Definite Hist. of Past Suppuration L.
Congestion	No nerve deafness	58.7	24.0	42.5	47.5	100	81.3	61.5	61.5
	With nerve deafness	73.0	73.0	67.0	79.0			66.7	83.3
Scars	No nerve deafness	0.0	0.0	12.5	10.0	54.5	54.5	53.8	61.5
	With nerve deafness	0.0	0.0	11.8	3.4			100	94.0
Retraction	No nerve deafness	58.7	53.0	60.0	72.5	72.7	72.7	53.8	38.5
	With nerve deafness	60.0	47.0	67.0	63.0			66.7	66.7
Adhesions	No nerve deafness	0.0	0.0	12.5	12.5	0.0	0.0	23.2	23.2
	With nerve deafness	0.0	0.0	4.2	4.2			16.7	16.7
Dim. Motion	No nerve deafness	12.5	18.5	52.5	47.5	54.5	54.5	61.5	38.5
	With nerve deafness	19.8	19.8	34.6	54.6			83.3	66.6
Calcium	No nerve deafness	30.0	24.0	35.0	37.5	63.6	54.5	30.7	30.7
	With nerve deafness	33.0	20.0	37.0	29.0			33.3	33.3
Pink Prom.	No nerve deafness	24.0	30.0	45.0	37.5	36.3	45.4	30.7	53.8
	?	30.0	24.0	15.0	15.0	18.2	9.2	7.7	0.0
	With nerve deafness	60.0	52.0	46.0	37.0			66.7	66.7
	?	0.0	0.0	0.0	0.0			21.0	21.0

it was by the presence or absence of scars that these differential diagnoses could be made.

Retraction was marked under all six subdivisions, being therefore not only coincident with detectable past otitis media.

Adhesions and diminished movement of the drum membrane or malleus were naturally most marked in the suppurative cases. Note that even in the nerve deafness cases with no detectable complications limited drum or malleus movements were seen. It is evident that these factors contributed to the hearing loss in many instances and really were evidences of past inflammation.

Calcium deposits usually near the umbo were particularly noted in the children. Can it be that in the adults some of these deposits may have been absorbed? In the adults they were present in both ears in about one-third of the cases throughout. In this connection it may be that what we called calcium deposits were in reality sometimes only very white fibrous tissue (or fat). Dense fibrous and lipiodol areas have a predilection for becoming calcified.

The incidence of pink glow upon the promontory was particularly high in the ears with nerve deafness. Fifty-two to 60 per cent in those without complications and 67 per cent in those with past suppuration. In the first instance may be because it was not obscured by thickening and in the last instance because it can be better seen through thin membranes filling in old perforations. In many instances it was questionable and therefore recorded only opposite the question marks. The glow is best observed with the aid of a daylight lamp and a pneumatic ear speculum so that the drum may be stretched and brought nearer to the promontory.

Roentgenograms show no severe involvements in the frontal or sphenoid sinuses with the exception of a few of the frontals in the masked and suppurative divisions. It is interesting to note that in every case with nerve deafness opposite no complications, and in from 67 to 83 per cent of the ears with definite past suppuration, there was one or two degrees of involvement in the frontals.

In the maxillary and ethmoid sinuses there was also a low percentage showing severe involvement, although in general the per-

THE DEGREES OF INVOLVEMENT OF THE NASAL SINUSES AS SHOWN BY STEREOSCOPIC ROENTGENOGRAMS IN THE THREE SUBDIVISIONS IN THE TWO MAIN CLASSES OF OTOSCLEROSIS. UNDER EACH DEGREE THE FIGURES INDICATE THE PERCENTAGE OF EARS INVOLVED.

No. of Cases		Degrees of Involvement in Nasal Sinuses									
		x		xx		xxx		xxxx		All Degrees	
	No Nerve Deafness—FRONTAL	R.	L.	R.	L.	R.	L.	R.	L.	R. Total	L.
17	No complications	53.0	47.0	12.4	18.5	0.0	0.0	0.0	0.0	65.5	65.5
40	Masked past inflammation	50.0	47.5	15.0	20.0	7.5	0.0	0.0	0.0	72.5	67.5
11	Masked past inflammation, children	63.6	63.6	18.1	18.1	9.1	9.1	0.0	0.0	80.8	80.8
13	History of past suppuration	38.5	38.5	23.0	46.1	7.7	0.0	7.7	0.0	76.9	84.6
	With Nerve Deafness										
15	No complications	60.0	46.0	40.0	26.0	0.0	0.0	0.0	0.0	100.0	72.0
24	Masked past inflammation	63.2	42.0	12.0	16.0	0.0	0.0	0.0	0.0	75.8	58.0
6	History of past suppuration	50.0	50.0	16.7	33.3	0.0	0.0	0.0	0.0	66.7	83.3
	No Nerve Deafness—SPHENOID										
17	No complications	24.0	18.0	0.0	0.0	0.0	0.0	0.0	0.0	24.0	18.0
40	Masked past inflammation	52.5	55.0	0.0	0.0	0.0	0.0	0.0	0.0	52.5	55.0
11	Masked past inflammation, children	27.2	36.3	18.1	9.2	0.0	0.0	0.0	0.0	45.3	45.5
13	History of past suppuration	23.2	23.0	15.3	7.0	0.0	0.0	0.0	0.0	38.5	30.0
	With Nerve Deafness										
15	No complications	26.0	26.0	6.0	6.0	0.0	0.0	0.0	0.0	32.0	32.0
24	Masked past inflammation	37.0	29.0	8.4	8.4	0.0	0.0	0.0	0.0	45.4	37.0
6	History of past suppuration	33.4	16.7	0.0	16.7	0.0	0.0	0.0	0.0	33.4	33.4
	No Nerve Deafness—MAXILLARY										
17	No complications	34.5	30.0	18.5	24.0	5.7	5.7	0.0	0.0	58.7	59.7
40	Masked past inflammation	32.5	37.5	22.5	27.5	10.0	10.0	0.0	0.0	65.0	75.0
11	Masked past inflammation, children	36.3	27.2	36.3	18.1	9.3	27.2	0.0	0.0	81.9	72.5
13	History of past suppuration	53.8	46.1	0.0	23.0	7.7	7.9	0.0	0.0	61.5	77.0
	With Nerve Deafness										
15	No complications	20.0	60.0	14.0	7.0	0.0	0.0	0.0	0.0	34.0	67.0
24	Masked past inflammation	33.6	33.4	33.6	16.8	4.2	12.6	4.2	4.2	76.6	67.0
6	History of past suppuration	33.3	16.7	33.4	16.7	0.0	0.0	0.0	0.0	66.7	33.4
	No Nerve Deafness—ETHMOID										
17	No complications	53.0	40.6	34.5	24.0	0.0	5.7	0.0	5.7	87.5	76.0
40	Masked past inflammation	50.0	30.0	25.0	27.5	0.0	0.0	0.0	0.0	75.0	57.5
11	Masked past inflammation, children	36.3	45.4	36.3	27.2	9.1	9.1	0.0	0.0	81.7	81.7
13	History of past suppuration	30.7	30.7	23.0	23.2	7.7	0.0	0.0	0.0	61.4	53.9
	With Nerve Deafness										
15	No complications	50.0	60.0	26.0	20.0	0.0	0.0	0.0	0.0	76.0	80.0
24	Masked past inflammation	37.8	42.0	33.6	25.0	8.4	8.0	0.0	0.0	79.8	75.0
6	History of past suppuration	66.6	33.3	16.7	33.3	16.7	0.0	0.0	0.0	100.0	66.6

centage of involvement was high. In the left antrum in children 27 per cent showed a moderately severe involvement. Attention is called to the fact that 33 to 67 per cent of antrums with nerve deafness with past histories of inflammation in the ears showed a mild or moderate involvement in the maxillary sinuses.

The maxillary and ethmoid sinuses on both sides of the head often seemed about equally involved in all types of complication, but the ethmoids under masked inflammations (forty cases) with non-nerve deafness show the right side was 20 per cent more frequently involved than the left.

Under the non-nerve division the highest percentage of involvement in the ethmoids are shown with the ears with no complications (88 per cent). It must be constantly borne in mind that unless I state to the contrary, reference is made to the percentage of sides and not of the cases. The latter naturally show a greater percentage.

The high percentage of nasal sinuses showing involvement suggests, even in the nerve deafness cases, that the hearing losses by bone and air conduction may be due, at least in part, to coincident lesions in the middle ears and not wholly to those in the nervous apparatus of hearing.

How do these roentgenologic findings compare with those in nonotosclerotic lesions of the ear? I can state that in spite of the high percentages in otosclerosis there is definitely a smaller percentage of involvement, both as to number and severity, in uncomplicated nerve deafness and a much greater percentage in recurrent and chronic otitis media. The exact variations I shall report at some future time.

THE DEGREES OF INVOLVEMENT OF THE NASAL SINUSES AS SHOWN BY STEREOSCOPIC ROENTGENOGRAMS IN 20 INDIVIDUALS WITH NORMAL HEARING.

No. of Cases		Degrees of Involvement in Nasal Sinuses									
		x		xx		xxx		xxxx		All Degrees	
		R.	L.	R.	L.	R.	L.	R.	L.	R. Total	L.
20	Frontal	40.0	40.0	15.0	20.0	0.0	0.0	0.0	0.0	55.0	60.0
	Maxillary	25.0	30.0	10.0	10.0	5.0	5.0	0.0	0.0	40.0	45.0
	Sphenoid	45.0	35.0	5.0	5.0	0.0	0.0	0.0	0.0	50.0	40.0
	Ethmoid	35.0	40.0	20.0	15.0	0.0	0.0	0.0	0.0	55.0	55.0

The important question is, Does the normal hearing patient have like evidences of past and present lesions in the nasal

sinuses? To determine this I have tabulated all my cases which were not in otosclerotic families and who had normal hearing at the time I examined them. These twenty may serve as controls, although they do not present a fair sample, because with three exceptions they had come to me suffering from some form of ear, nose or throat trouble.

Note that both the degrees and the percentages of involvement in the nasal sinuses are low as compared with the otosclerotic cases. Almost all the totals are made up from the one degree involvements. It is surprising and illuminating to find these controls comparatively free from the involvements present in the otosclerotic cases, although many of them had suffered acute otitis media at some time in their lives. Evidently their recovery was complete.

The almost universal evidence of past inflammation in the nasal sinuses in the otosclerotic cases and the comparative freedom from inflammatory signs in the controls still further strengthens the argument for inflammatory middle ear lesions as etiologic factors in the deafness accompanying otosclerosis, and, in fact, as a factor in the etiology of otosclerosis itself.

Without nerve deafness from 47 to 82 per cent, and with nerve deafness from 17 to 66 per cent showed some degree of absorption had taken place in the right or left ethmoid septa. With masked past inflammations this ran up on one side of the head or the other to 23 or 25 per cent under every heading, except the xxxx. It is curious that opposite no complications one degree of absorption showed nil for the right sides and 40 per cent for the lefts.

With nerve deafness the percentages of absorption are generally smaller on both sides than with non-nerve deafness. Note particularly that there was no absorption in the definite past suppurative cases on either side under x and xx and xxxx.

The presence of absorption indicates that some inflammatory process has caused the lime salts to disappear from the ethmoid septa. It is an indication of past inflammation in the sinuses.

Condensing osteitis was noted in but three instances under xxx and none under xxxx.

THE DEGREE OF ABSORPTION, CONDENSING OSTEITIS, AND EDEMA IN THE BONY ETHMOID SEPTA AS SHOWN BY STEREOSCOPIC ROENTGENOGRAMS IN THE THREE SUBDIVISIONS IN THE TWO MAIN CLASSES OF OTOSCLEROSIS. UNDER EACH DEGREE THE FIGURES INDICATE THE PERCENTAGE OF RIGHT AND LEFT SIDES INVOLVED.*

No. of Cases	ABSORPTION	x		xx		xxx		xxxx		All Degrees	
	No Nerve Deafness	R.	L.	R.	L.	R.	L.	R.	L.	R. Total	L.
17	No complications	0.0	40.0	34.5	18.5	13.5	11.5	0.0	0.0	47.0	70.0
40	Masked past inflammation	15.0	22.5	22.5	17.5	20.0	25.0	0.0	0.0	57.5	65.0
11	Masked past inflammation, children	36.5	18.2	27.3	18.2	9.1	18.2	9.1	0.0	82.0	54.6
13	History of past suppuration	15.4	23.1	30.7	30.7	23.2	7.7	0.0	0.0	69.3	61.5
	With Nerve Deafness										
15	No complications	20.0	26.0	26.0	26.0	6.0	0.0	0.0	0.0	52.0	52.0
24	Masked past inflammation	16.0	9.4	33.6	25.2	12.6	8.4	4.2	0.0	66.4	43.0
6	History of past suppuration	0.0	0.0	0.0	0.0	16.7	16.7	0.0	0.0	16.7	16.7
	CONDENSING OSTEITIS										
	No Nerve Deafness										
17	No complications	11.5	11.5	11.5	18.5	0.0	0.0	0.0	0.0	30.0	30.0
40	Masked past inflammation	15.0	12.5	12.5	10.0	0.0	5.0	0.0	0.0	20.0	17.5
11	Masked past inflammation, children	9.1	38.6	9.1	0.0	0.0	0.0	0.0	0.0	18.2	36.6
13	History of past suppuration	7.7	7.7	7.7	7.7	0.0	0.0	0.0	0.0	15.4	15.4
	With Nerve Deafness										
15	No complications	6.0	13.0	6.0	6.0	0.0	0.0	0.0	0.0	12.0	19.0
24	Masked past inflammation	21.0	21.0	4.0	8.0	0.0	0.0	0.0	0.0	25.0	29.0
6	History of past suppuration	0.0	0.0	0.0	16.7	16.7	16.7	0.0	0.0	16.7	33.4
	EDEMA										
	No Nerve Deafness										
17	No complications	12.5	24.0	53.0	34.5	0.0	11.5	0.0	0.0	65.5	70.0
40	Masked past inflammation	30.0	37.0	27.5	35.0	15.0	0.0	0.0	0.0	72.5	72.5
11	Masked past inflammation, children	9.1	18.2	45.5	54.5	9.1	9.1	0.0	0.0	63.7	81.8
13	History of past suppuration	15.4	7.7	53.8	30.7	0.0	30.7	0.0	0.0	69.2	69.1
	With Nerve Deafness										
15	No complications	33.0	33.0	46.0	33.0	6.0	20.0	0.0	0.0	85.0	86.0
24	Masked past inflammation	25.2	29.4	37.0	37.0	12.6	8.4	0.0	0.0	74.8	74.8
6	History of past suppuration	0.0	16.7	33.3	33.3	16.7	0.0	0.0	0.0	50.0	50.0

*x equals mild, xx equals moderate, xxx equals moderately severe, xxxx severe involvement.

STEREOSCOPIC ROENTGENOLOGIC FINDINGS IN THE EAR BONES IN THE TWO MAIN CLASSES OF OTOSCLEROSIS AND THE THREE SUBDIVISIONS UNDER EACH OF THESE.

	No Other Complications		Masked Past Inflammation				Definite History of Past Suppuration	
			Adults		Children			
PNEUMATIC MASTOID	R. L.	R. L.	R. L.	R. L.	R. L.	R. L.	R. L.	R. L.
No nerve deafness —large	82—76		85—76		62—82		69—69	
small		6—13		10—13		18— 9		16—23
With nerve deafness—large	93—93		76—76				67—50	
small		0— 0		16— 9				0—17
SCLEROTIC MASTOID								
No nerve deafness	13—13		13— 5		0— 0		23—16	
With nerve deafness	6— 6		8— 8				17— 0	
INFANTILE—Six Cases								
CORTEX								
No nerve deafness —thick	30—30		40—30		46—55		23—31	
thin		70—70		50—58		55—55		69—62
With nerve deafness—thick	26—33		46—50				50—33	
thin		67—60		46—37				50—50
ZYGOMA CELLULAR								
No nerve deafness	59—59		40—38		46—37		54—54	
With nerve deafness	60—66		37—33				50—50	
SEPTA								
No nerve deafness —thick	37—47		55—53		37—37		34—46	
thin		41—35		33—28		63—55		15—15
With nerve deafness—thick	46—46		55—63				33—17	
thin		40—46		29—16				33—50
LUMEN								
No nerve deafness —large	23—18		15— 8		18—18		31—31	
small		47—53		45—35		55—46		69—62
With nerve deafness—large	46—46		55—63				33—17	
small		40—46		39—16				33—50
AUDITORY CANALS								
No nerve deafness —large	47—47		55—60		73—73		23—23	
small		35—35		25—23		9— 9		69—69
With nerve deafness—large	48—48		67—59				50—17	
small		52—52		29—38				17—33

PETROUS—In no instance was there any evidence of disease in the petrous tip.

Edema of the ethmoid bony septa was present with non-nerve deafness ears in from 64 to 82 per cent on one side or the other. In nerve deafness ears in from 50 to 86 per cent on one side or the other. The two sides show, generally speaking, an equal percentage with edema of the ethmoid septa. Strange to note, the nerve cases show a greater percentage with edema in the ears opposite no complications. This indicates that although the edema of the bony septa continues there need be no visible signs of present or past ear infections. It does not mean that such have not taken place.

Large pneumatic mastoids were found in 69 to 85 per cent of the ears in the cases without nerve deafness and in 50 to 93 per cent of the ears in cases with nerve deafness in the various groups. The mastoid cells showed thick and thin walls in varying proportions. This is recorded for future reference, as it has no apparent connection with otosclerosis. It simply shows that the large pneumatic mastoid with small cells about the antrum is the prevailing type. The lumen of the cells around the antrum showed small in a great majority of cases with no nerve deafness and with nerve deafness about an equal percentage large and small, except with the past marked inflammations, where the large cells were present in 55 to 63 per cent of the ears.

I do not know what, if anything, these variations may signify.

Absorbed areas and osteitis show so rarely that I have omitted them from the table.

It appears that although Dr. Law and I expected something definite to show from our mastoid and petrous roentgenograms, we have been disappointed, and in the future shall alter radically our technic. One must constantly keep in mind, however, that both X-ray and clinical pictures change, and unless both are observed during the heights of pathologic activity the lesions may be missed.

The blood count, differentials and blood volumes present nothing of significance except that they are within the normal. The blood chemistry, in so far as cholesterol, calcium, phosphorus and sugar are concerned, also appear to be within normal limits. This was my finding in fifty-eight cases of otosclerosis reported three

BLOOD COUNTS, DIFFERENTIALS, AND BLOOD CHEMISTRY IN THE TWO MAIN CLASSES OF OTOSCLEROSIS AND THE THREE SUBDIVISIONS IN EACH OF THESE.

In every instance the figures represent the average.

No. of Cases		Hgb	Red Cells	White	Ply.	S.	L.	Vol.	Chol.	Calc.	Phos.	Sug.
	NO NERVE DEAFNESS	%			%	%	%	%	in grams per 100 cc.			
17	No complications	75	4,224,000	7,200	54	38	5.5	37	151.6	9.3	3.4	93.0
40	No history past inflammation	80	4,450,000	7,450	57	39	4.5	34	151.0	9.3	3.0	95.0
11	No hist. past inflam., children	77	4,400,000	7,690	57	37	5.0	40	157.0	10.0	3.5	89.0
13	History past suppuration	81	4,300,000	7,900	58	35	4.3	41	163.0	9.0	3.0	99.0
	WITH NERVE DEAFNESS											
15	No complications	76	4,630,000	8,300	74	40	4.0	40	114.0	9.0	3.1	93.0
24	No history past inflammation	81	4,650,000	7,420	59	43	4.3	43	177.0	9.0	3.0	106.0
6	History past suppuration	75	4,220,000	8,000	51	25	6.2	37	160.0	9.7	3.3	99 3

Wassermann was positive in but a single instance.

years ago before this Society,[5] and furnishes confirmation for the former determination. It may be noted that in the children the serum calcium and phosphorus was a little higher normal than the adults, as was to be expected.

Many items have been omitted from the table given above because they also showed no variations from normal.

The absence of changes in the blood chemistry in established otosclerosis (both static and progressive) indicates at least that the process persists in spite of normal blood calcium, phosphorous and cholesterol. It does not mean that at the start of the otosclerotic foci some changes in the blood salts or chemistry was not present.

It is, however, logical to assume that the blood chemistry has little or nothing to do with the continuation of the otosclerotic process because otherwise in the active cases some abnormalities should be found. This indirectly focuses attention upon the local lesions in and about the sclerotic foci, and especially upon the inflammatory lesions so often found in the middle ear.

Someone will ask, if otitis media is so prevalent, and a factor in the etiology of otosclerosis, why does otosclerosis not occur more frequently. The answer is that it does occur often but is not diagnosed. Routine autopsies give an incidence for otosclerosis of more than 1 in 20 in all individuals, though few of these show bony ankylosis. This suggests that in routine autopsies in patients with deafness the incidence of otosclerosis would be much higher. Otosclerosis pathologically is not a rarity, otosclerosis clinically is a comparative rarity only because of failure of diagnosis.

Studies of microscopic sections confirm the clinical observations. There are in our collection very few cases which show no evidence of inflammation. The typical finding is a thickened mucoperiosteum, and often calcification of connective tissue, somewhere in the middle ear cavity. Most usually this occurs in the anterior part of the cavity, above the entrance of the eustachian tube, and about the tensor tympani muscle. (Fig. 1.)

These processes seem to be more prominent in three types of cases. (1) In cases with a history of chronic ear or sinus dis-

ease; (2) in cases of otosclerosis (Fig. 2); (3) in cases with various high tone losses.*

They are by no means confined to these types. They seem, however, to be less common and less marked in other cases.

The above statements are derived from a study of five cases of otosclerosis from our own collection in the Department of Pathology at Columbia; 14 cases of otosclerosis presented to us by Professors Wittmaack, Mayer, VonEicken, Ruttin, Brunner, and

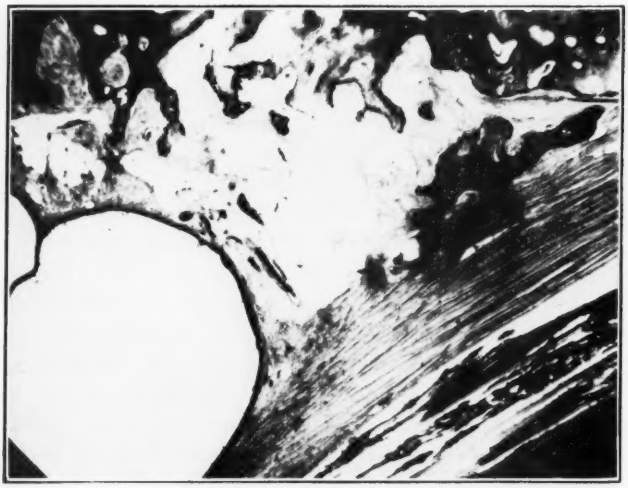


Fig. 1. Past masked inflammation. Fat, dense connective tissue and spicules of bone surrounded by calcified connective tissue—in region of tensor tympani muscle—typical finding of more or less severity in all individuals.

now at the Central Bureau of Research, and 34 cases of otosclerosis from the collection of the Johns Hopkins Hospital.†

*These cases are being prepared for publication and will be reported by Drs. Guild, Crowe, Polvogt, etc.

†We wish to thank the members of the Laboratory for Otological Research at Johns Hopkins for their generous co-operation in opening their files to us. Only through the study of their clinical histories, audiograms, and serial sections were we able to study a sufficient number of specimens to be certain of the great prevalence of masked past inflammations in otological material.

For controls we used our entire collection of some 160 bones and a series of 20 consecutive cases studied in the Johns Hopkins collection. These all show some dense fibrous tissue or calcified connective tissue, suggestive of actual or masked past inflammation. This means that middle ear disease is an extremely common

Fig. 2. Otosclerotic focus in typical site. Note peninsula of fibrous tissue running from middle ear cavity into the focus: Extent of fibrous tissue loading of anterior crus of the stapes; blood vessels running deep into bone from middle ear.

disease; much more common than is generally supposed. It casts considerable doubt on the belief that inflammatory processes are rare in otosclerotic cases. Every otologist knows how often it is impossible or difficult to diagnose middle ear disease even when its presence may be subsequently quite apparent. The following

Fig. 3. Past masked inflammation. Left mastoid cells of case which had severe mastoiditis on the right.

illustrates a case which came to autopsy. Fig. 3 is the mastoid process of the left ear of a patient who was watched for three weeks with middle ear disease and eventually mastoiditis on the right side. During this time there was never any evidence of inflammatory lesions in the left drum. The patient died from a brain abscess secondary to petrositis on the right and showed considerable evidence of bony regrowth in the petrosa and a chronic

mastoiditis on the left which was never made out clinically either by X-ray or otoscopic examination.

It may well be, that the inflammatory episode which seems to be so common in all individuals is a factor of great importance in the individual predisposed to otosclerosis; both Fraser[2] and Wittmaack's[3] theories may be right in principle. Certainly we know that when there is an inflammation, small thromboses often occur in the veins draining the inflamed tissues.[4] Is there any reason why these may not be etiologic factors for the venous stasis claimed by Wittmaack? The answer has heretofore been that evidence or history of inflammation is rarely forthcoming. I have furnished such evidence from clinical data and now shall do so from pathologic data.

I am inclined to agree with Fraser that practically everyone at one time or another has harbored inflammation in his ears just as surely as everyone has experienced head colds. The blood vessels which supply and drain the site of predilection for otosclerosis run superficially over the promontory and anastomose with those in the labyrinthine wall. (Fig. 2.) It is really surprising that thromboses are not found in them more frequently; perhaps they do occur but the vessels being very small they are quickly recanalized or replaced and years later one cannot see or recognize the signs of former blocking unless there takes place recurrent inflammatory lesions to recreate or form new and obvious thrombi.

Pathologically there is in certain instances another fact which points to an infectious origin of otosclerosis, namely, dense fibrous scar tissue which often extends from the middle ear cavity into the focus. (Fig. 2.) We have not been able to uncover a case of osteomyelitis at the common site for otosclerosis, namely the anterior border of the oval window, but we have found areas of absorption and bone regrowth in the footplate of the stapes which may be of significance because otosclerosis occurs here either alone* or in conjunction with foci at the point of predilection.

Another important point is demonstrated in our studies. It is found that in a large number of cases of otosclerosis there are

*Cases to be reported.

fibrous adhesions between the otosclerotic focus and the anterior crus of the stapes, whether there was a bony ankylosis or not. (Fig. 2.) In the few cases where an audiogram was available this fibrous adhesive process produced a diminution of hearing of 15 to 20 S. U. over the entire scale. Evidently this loss would have been superimposed upon any loss due to calcium deposits in the annular ligament. This deafness was sometimes ascribed by the examiner to inattention, but we believe he was in error as no such consistent losses have been found without cause in our audiometric tests even in the very sick or in those given to inattention. Even extremely sick patients cooperate with the examiner and rarely is the normal found more than 10 S. U. down in a fairly noisy ward.

SUMMARY.

The clinical evidence indicates that in these cases of otosclerosis there was definite evidence of the almost universal prevalence of past inflammatory lesions in the head spaces, particularly in the nose and middle ear, and that there were no blood abnormalities of any kind which could be linked up with the otosclerotic process. The roentgenograms of the sinuses strongly strengthen the clinical evidence. Syphilis may also be excluded because the Wassermann reaction was positive in but a single instance. The pathologic evidence substantiates the clinical observations.

I could add to the clinical and pathologic evidences from material gathered since the compilation of these statistics, but sufficient has been produced from the preliminary studies to reach the following conclusions.

CONCLUSIONS.

"Progressive deafness" is not synonymous with otosclerosis because other lesions often cause progressive deafness.

Otosclerosis may not produce progressive deafness because otosclerosis frequently causes no ankylosis of the stapes footplate and no deafness. All the deafness accompanying otosclerosis may not be due to bony ankylosis because there are usually accompanying complications which are capable of causing deafness.

Often all the deafness accompanying otosclerosis in certain cases may be due and has been found to be due to fibrous adhesions. Conceivably all the deafness accompanying otosclerosis may be due to a nervous lesion and have no obstructive background whatsoever. Examiners should keep in mind the fact that otosclerosis may and usually does occur coincidently with other lesions of the ear.

The detection of other lesions does not preclude a subsequent diagnosis of otosclerosis.

The negative history of other lesions does not exclude their presence at some time in the life of the patient.

The study of the histories and the clinical, roentgenologic and pathologic data all give evidence of the almost universal presence of lesions in the nose and ear spaces indicative of past inflammatory reactions, and these evidences are not much less than in cases with definite evidence of suppuration.

The only approach to treatment of otosclerosis which promises prevention or arrest of the lesions is treatment to combat the tendency to recurrent or chronic inflammatory processes in the nose and ear spaces, particularly the nasal sinuses, because it is herein that the foci of infection tend to linger, ready to resume again and again an active state and so threaten or continue the inflammations, suppurations, adhesions, thrombosis and ankylosis in the middle ear.

The inflammatory processes are not to be ignored as factors in the deafness accompanying otosclerosis and in its etiology.

The diagnosis of early otosclerosis may be, and frequently is, dependent upon other lesions causing obstructive deafness, which possibility must be kept in mind.

Especially is the otologist cautioned to search for otosclerosis even though his immediate diagnosis is definitely a residual inflammatory lesion of the ear, because otosclerosis is usually accompanied by evidences of such lesions in the past.

Treatment of the complications of otosclerosis is not only indicated as the only management which may improve the deafness

but as the only management which at present holds out any hope of preventing or arresting the otosclerotic process.

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LVI.

A STUDY OF TWENTY-FOUR CASES OF NECK INFECTION.*

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Medical history records that many a physician's desire to know more about a given disease has been aroused and stimulated by one or more experiences with patients having that disease, particularly if the outcome of some has been disastrous. This paper is the result of such experience. I believe that the subject is but indifferently understood by the rank and file of the profession, and sincerely trust that by submitting the following findings I may arouse an appreciation, the while leaning very heavily on the teachings of master thinkers, and disclaiming any originality. The textbooks leave much to be desired after perusal. As a result of study and experience, phenomena which were at first incomprehensible or overlooked will often become clear and easily recognized. No locality of human habitation in which there occur infections of the upper respiratory tract is exempt from these neck infections. All ages and both sexes are victims. Much suffering can be ameliorated and many fatalities prevented. Physicians and surgeons practicing in small communities, and whose work is done in small hospitals will encounter a surprisingly large number of these cases.

Analysis of the case histories which form the basis of this paper has revealed much of which I was previously ignorant. Local symptoms and signs can be explained by the applied anatomy, and where understood and recognized indicate the site of the infection and its remedy. Fatal septicemia can often be prevented. Thrombosis of the internal jugular vein is one of the most fatal complications of tonsillar and pharyngeal infections. It may be associated with frank pus in the throat, or the throat

*Presented before the American Academy of Ophthalmology and Otolaryngology, Montreal, September 19-23, 1932.

infection may have been apparently slight, and may have entirely cleared up. After any throat infection, light or severe, in which a septic temperature persists, thrombosis of the internal jugular vein should be ruled out. The management of deep pus in the neck requires, on the part of the operator, courage and decision. A knowledge of the applied anatomy of the neck makes the search for pus much surer. In spite of the fact that attention was called to this subject many years ago, cases still go on to fatal termination unrecognized. Recent cases have shown that not only the laryngologist fails to recognize this condition, but that the general surgeon is equally ignorant of it. The fact that the laryngologist is frequently called in consultation by the family physician or general surgeon makes knowledge of the subject obligatory.

The purpose of this paper is first to discuss infections of the neck, especially deep pus in the neck. The source of such infection is, as Mosher has said, "in or about the tonsils." In connection with deep pus in the neck there is often an unrecognized thrombosis of the internal jugular vein which gives a fatal septicemia. The second purpose of the paper is to discuss the surgical route to deep pus in the neck. Mosher,[1] in 1920, discussed thrombosis of the internal jugular vein from infection from the tonsils. Later[2] he discussed the surgical approach to deep pus in the neck, and outlined a definite plan of approach.

The material for this paper is based on an analysis of twenty-four cases of infection of the neck, all except two of which were treated operatively. All of the pharyngomaxillary cases, comprising 58 per cent of the total, came to me unrecognized or with a tentative diagnosis of quinsy sore throat. The severe infections which are caused by furuncles about the face and neck are not included in this series.

INCIDENCE OF AGE AND SEX.

There were fourteen females (58 per cent) and ten males (41 per cent). Among the adults and adolescents (ages 14 to 53) there was a majority of females, eight to four. Among the infants and children (ages 8 months to 7 years), six of each sex.

Five cases under two years of age.....20+ %
Seven cases between ages of two years and seven years.....29+ %
Four cases between ages of fourteen and nineteen years.....16+ %
Eight cases between ages of twenty and fifty-three years.....33+ %

No startling conclusions are drawn from the factors of age and sex. They may be summarized as follows:

Infancy and childhood	50%
Adolescents and adults	50%

ETIOLOGY.

This is considered from the standpoint of the part through which the infection enters, the portal of entrance, and any known pre-existing disease or injury, contributory or provocative. The determination, by history and examination, of the exact portal of entrance should be given important consideration, for with knowledge of this it may be possible to anticipate the course of the infection when invasion of the cervical region has taken place, and gives valuable information as to the treatment, medical and surgical, best adapted to the case. For instance, if the infection of the neck results from infection of the teeth in the mandible, the submaxillary and submental regions will probably be the first part of the neck involved.

Analysis of the etiology in these cases, few though they be, definitely corroborates what Mosher has stated, which is, that the greatest single cause of these infections is "in or about the tonsils." Next in frequency comes the pharynx. Acute or subacute inflammation of the tonsils preceded or accompanied the attack in eleven cases (45 plus per cent). In several the tonsillitis occurred from one to two months prior to the outbreak in the neck. Lymph node inflammation was believed to be the connecting link in these. Inflammation of the pharynx alone accounts for four cases. In these the tonsils had been removed from one to three years previously. Tonsil remnants located in much deformed and scarred tonsil fossæ are to be viewed with suspicion. It may not be possible to definitely determine whether the infection enters through the tonsil or by way of the pharyngeal wall, as where there is tonsillar inflammation there is also present some pharyngeal inflammation, and vice versa. All of the mucosa of the upper respiratory tract may be inflamed, including that of the nasal accessory sinuses. The infection may begin in the nose and sinuses, and be followed by involvement of the pharynx and tonsils. Pharynx and tonsil inflammations account for fourteen cases (58 per cent). Two of these followed surgical trauma.

Three cases were of dental etiology (12 per cent). They were all mandible in location, two following extractions, and one necessitating multiple extraction at the time of operation because of extensive alveolar abscess. The remainder (29 per cent), numbering seven cases, exhibits as many varieties of etiology as there are cases. Enumerated, these are as follows:

1. One, case No. 19, followed a tonsillectomy, the infection entering through one tonsil fossa, and remained localized.

2. One, case No. 12, followed surgical removal of an adenoid. This involved the lateral area on one side of the pharynx, the process having no resemblance to the typical retropharyngeal abscess.

3. One, case No. 14, was caused by infection induced by the injection of a local anesthetic along the posterior surface of the maxilla in the direction of the sphenopalatine fossa.

4. One, case No. 4, followed trauma to the mucosa of the hypopharynx by a bolus of food containing an unmasticated bread crust.

5. One, case No. 15, began in an infected lymph node lying in contact with the fascia of the submaxillary salivary gland. It was never possible, by history and examination, to determine the exact point of entrance of the infection invading this lymph node.

6. One, case No. 21, is regarded as having been caused by infection of the capsule of the thyroid gland. It is concluded that the infection may have begun in a lymph node lying in contact with this fascia. When first seen the case was fairly well along, and had an appearance strikingly like an ordinary goiter. There were no symptoms indicating derangement of the function of the thyroid. When first seen there was distinct fluctuation.

7. One, case No. 13, after careful consideration, is attributed to inflammation of the petrous part of the temporal bone. It could not be proven by autopsy findings because the patient got well. At the time, I talked with Dr. Wells P. Eagleton over the telephone about this case, and he advised drainage in the neck before attempting an attack on the bone. This was done, with gratifying results. The possibility that the pharyngeal infection in this case may have resulted from extension along the auditory tube from the middle ear has been considered. Radiograph of

the base, vomiting, and first branch trigeminal pain point strongly to the petrous. There was at no time any paresis of the sixth nerve. Separated by a long interval of time there had been done two mastoid operations.

It is to be noted that in this group of cases there are none caused by the following conditions: External neck injuries, disease of the cervical vertebræ, Bezold's abscess, fractures of the larynx and trachea, perforation of the esophagus, suppurating cysts, fracture and injuries of the jaw.

The bacteriology of these cases was in no sense complete, but enough is in evidence to give basis for one conclusion, viz., that at least 50 per cent of these infections are caused by the hemolytic streptococcus.

No culture obtained	six cases
Organisms failed to grow	two cases
Pure culture streptococcus hemolyticus	ten cases
Streptococcus, not typed	two cases
Staphylococcus, strepto and pneumococcus, not typed	one case
Strepto and staphylococcus, not typed	one case
Streptococcus viridans and staphylococcus albus	one case
Staphylococcus aureus	one case

APPLIED ANATOMY.

A familiarity with the applied anatomy of the cervical fascia is indispensable to a comprehension of the problems arising from infection in this region. What is here said about applied anatomy is based entirely on the work of Mosher, and deserves great emphasis.

There are three large fascial compartments in the upper part of the neck which communicate more or less directly with the sheath of the great vessels. These are the parotid, the submaxillary and the pharyngomaxillary. The parotid and submaxillary compartments contain the respective salivary glands, and require no detailed description except a mention of the fact that the retromandibular process of the parotid gland is devoid of fascia at its inner extremity where it lies in the most intimate relationship with the pharyngomaxillary space. While the submaxillary gland itself is separated from the mucosa of the floor of the mouth by a muscle layer, it does get a direct communication with the mouth cavity through its duct. The pharyngomaxillary space is somewhat cone-shaped, the apex of the cone being continuous down-

ward into the neck as the sheath of the great vessels, and its base uppermost at the base of the skull. By using a bit of imagination these three compartments may be likened to a trifolium, the sheath of the vessels being the stem, the three spaces the leaves.

The prevertebral fascia requires special mention because of the frequency of infection. No detailed description is necessary except to mention that it encircles the vertebral column and the muscles attached to it. Infections here have a decided tendency to extend rapidly downward. I have never observed a true midline retropharyngeal abscess to invade the pharyngomaxillary space. Perhaps this is also the explanation of the prompt recovery after incision through the mouth in cases not seen late. The applied anatomy when viewed as follows, explains these observations. See also Fig. 11.

High up in the neck the scaleni muscles, at their origins, lie in contact with the vertebral column. Lower in the neck these muscles course forward and laterally to reach their insertions on the first and second ribs. As seen from the front there is thus formed a pyramidal space, with its apex up at the skull base, its posterior side formed by the vertebral column and longus colli muscles, and the lateral sides by the scaleni muscles. Infections starting high up in the median line therefore would have a natural tendency to extend downward along the longus colli fascia rather than in a lateral direction. Infections which are lateral as well as midline at the start could extend laterally as well as downward, and quickly reach the vascular sheath which crosses the scalenus anticus obliquely.

A true midline infection would therefore extend into the posterior mediastinum along the vertebral column all the way. The lateral infection would travel forward along the scaleni muscles under the clavicle and into the costo-caracoid region of the chest wall, into the axilla, and also possibly into the posterior cervical triangle, where it would become superficial. It can be assumed therefore that if the infection takes this lateral course it has been in a lateral situation from the start, and should be differentiated from a true midline retropharyngeal abscess.

One other layer of fascia is of importance from the standpoint of applied anatomy. It has been given various names ac-

cording to locality. This fascia encircles the visceral space of Tillaux,[3] and is therefore well named the visceral layer. In the lower part of the neck it is also known as the pretracheal layer, and surrounds the trachea and esophagus. It splits anteriorly to envelop the thyroid gland. In the upper part of the neck it is known as the buccopharyngeal fascia, and covers the constrictor muscles of the pharynx and the buccinator. This is the fascia which is the seat of involvement when infections penetrate from the hypopharynx and esophagus. I earnestly recommend the reading of Mosher's description of the applied anatomy of the cervical fascia as published in the Transactions of this Society for the year 1929.

REGIONAL MANIFESTATIONS.

The following grouping of fascial plane involvement is an attempt at classification, and is suggested by the findings. It should be obvious that in these severe inflammatory conditions the involvement does not remain strictly confined to one particular fascial compartment.

1. Pharyngomaxillary space infection ..... 14 times—58 %
2. Submaxillary space infection ..... 5 times—20+ %
3. Carotid sheath infection ..... 5 times—20+ %
4. Cervical lymph node suppuration ..... 3 times—12+ %
5. Prevertebral fascia infection ..... 3 times—12+ %
6. Buccopharyngeal, pretracheal or visceral fascia infection 2 times— 8+ %

PHARYNGOMAXILLARY SPACE INFECTION.

From this summarized list it will be seen that infection in the pharyngomaxillary space is frequent, over half of the cases. Even in those where the beginning of the infection is distant from this space, as where the primary manifestation is in or near the submaxillary space, there will sometimes occur extension of the infection into the pharyngomaxillary space. In fact, it should be anticipated, and daily inspections of the pharyngeal wall made. Involvement should be considered present if there is trismus and swelling of the lateral pharyngeal wall with or without change of color. With the swelling of the lateral pharyngeal wall there is often what I have chosen to designate as a prolapse of the tonsil and palatal arch, which is in reality a displacement toward the

median line of the mouth. This prolapse or displacement can and does occur without the slightest swelling or enlargement of the tonsil itself, and helps to differentiate it from a peritonsillar abscess in which inflammatory swelling of the tonsil is present. When this fullness of one side of the palate is present the uvula has been seen to point almost horizontally toward the opposite side. In some cases there is very little, if any, inflammatory reaction to be seen with the swelling, but in other cases it is present. If present it must be differentiated from peritonsillar abscess, and this is not always easy. Swelling of the lateral pharyngeal wall is an aid in the differentiation, but is sometimes hidden by the palatal or faucial swelling. If there is swelling over the region of the parotid gland it may be considered additional evidence of deep infection. Where the infection is lower in the neck below the level of the angle of the jaw it does not involve this space, and these signs would not be present. When the pharyngomaxillary space is the seat of the initial invasion in the cervical fascia, that is to say, not the result of extension from one of the other compartments of the fascia the etiology is most frequently infection in the tonsils and pharynx. Infection in the nose, sinuses, adenoid, pharyngeal lymph nodes, cervical vertebræ, mastoid and petrous temporal bone are all possible primary sources.

Inflammations in the parotid and submaxillary spaces not rarely extend into the pharyngomaxillary space. There may be infection in the pharyngomaxillary space without any visible evidence of infection in the parotid or submaxillary spaces. Inability to open the mouth widely is one of the earliest signs of infection high up. Infection severe enough to cause phlebitis of the jugular vein early may exist without visible external swelling of the neck. If not drained such cases will go on to jugular thrombosis and die of septicemia. Infection outside of the tonsil remains a peritonsillar abscess so long as it does not penetrate the muscle bed forming the tonsil fossa. As soon as the infection penetrates the pharyngeal constrictor muscle it becomes pharyngomaxillary. This penetration may take place directly or through the veins, as was beautifully shown by Waldapfel[4] at the meeting of this Society in 1928.

SUBMAXILLARY FASCIA INFECTION.

This group, comprising the so-called cases of Ludwig's angina, may be subdivided into two groups, which, however, manifest themselves similarly. One group is of dental origin and the other is not. The cause of this latter group is sometimes obscure. In two of the cases it was attributed to lymph node inflammation. Though there were none in this series it is to be expected that infection in the floor of the mouth may be considered as a starting point. It is of significance, however, that the only cases in this series which had swelling and induration of the floor of the mouth were of dental origin. The inflammation rarely remains confined to the submaxillary space, but extends into the parotid space or into the pharyngomaxillary space or both, and downward below these into the lower part of the neck along the vascular sheath. The applied anatomy of the fascia of the submaxillary triangle region suggests that infection would find a barrier to direct downward extension at the hyoid bone where the fascia gets a firm attachment. For this reason deflection laterally to the vascular sheath is to be expected if extension is not checked. This observation is borne out clinically. It is noteworthy how frequently swelling in the submaxillary space becomes the initial manifestation of infection in the neck. Those of dental etiology yield promptly to submaxillary drainage, and can usually be given a good prognosis. In these trismus is extreme, and the floor of the mouth is usually involved. Those not of dental etiology are an insidious and dangerous type, and there being no primary involvement high up, swelling of the lateral pharyngeal wall of the naso- and oro-pharynx and trismus would not be present. If later there is extension into the pharyngomaxillary space these signs will appear. These cases should be drained early, and if sepsis continues and chills occur the carotid sheath should be immediately exposed. If blood cultures are positive ligation or resection cannot be too quickly done, for thrombophlebitis in distant veins may occur early. If sepsis characterized by wide fluctuations of temperature persists with negative blood cultures, ligation or resection is nevertheless indicated. This is particularly so if there are chills. All of these manifestations have occurred in cases in this series.

CAROTID SHEATH INFECTION.

This, as can be explained by the applied anatomy, is secondary to infection of any or all of the other compartments and their contained lymph nodes. When the infection begins in the submaxillary or parotid compartments it may extend directly to the sheath of the vessels. It may, however, extend to the pharyngomaxillary space first, the infection of the sheath becoming a later development. A tympanomastoid infection may intervene, and in turn cause a sigmoid sinus thrombosis. Tenderness on palpation over the carotid sheath and deep to the sternomastoid muscle is an unreliable sign of vein infection; so also is swelling; for both may be caused by inflamed lymph nodes, and not necessarily by inflammation of the vein wall. One should be guided more by the degree of sepsis than by these local signs, and absence of local signs should not be construed as sufficient reason to rule out the possibility of jugular sheath infection. Likewise their presence cannot be made the sole reason for jugular surgery.

There were five cases of carotid sheath infection, two of which did not go on to thrombosis. One of these, case No. 15, disclosed a peculiar fibrinous exudate on the sheath when it was exposed, and because I did not at that time realize the significance, the vein was not resected. After a stormy course, and aided by blood transfusions, the patient recovered. The other, case No. 12, is the one from which the photomicrograph was made, which shows the polynuclear infiltration of the vein wall. Blood cultures were negative in both of these. In one of the three cases in which there was thrombosis, case No. 16, the thrombosis occurred in the sigmoid sinus and was caused by a coincident tympanomastoid infection. In this the blood culture was positive. In the other two cases the thrombosis was caused directly by the neck infection. One of these, case No. 8, was one of my earliest cases, and was draining profusely through an incision in the lateral pharyngeal wall. Because of this and the marked subsidence of the swelling of the neck, it was concluded at consultation with general surgeon and another laryngologist that external drainage was not necessary. The patient died of sepsis caused by jugular vein and cavernous sinus thrombosis. In the other, case No. 24, the carotid

sheath infection was secondary to the submaxillary and parotid space infection, and after obstructive thrombosis had taken place the blood culture which had been positive became sterile. It is urged therefore that blood cultures be made early in the course of the disease because of the possibility that negative results will be obtained after the vein is corked off by the thrombus. The patient who died, case No. 8, gave negative blood cultures, but they were done late in the disease, and the negative results can now be explained on this basis. Blood cultures are most apt to be positive if the blood is taken during or immediately after a chill.

It cannot be too strongly emphasized that phlebitis of the jugular vein can take place early. Metastatic abscesses are to be expected when thrombosis occurs.

CERVICAL GLAND ABSCESSES.

The superficial glands which lie between the superficial fascia and the superficial layer of the deep fascia, drain into the glands of the deep fascia. Suppuration of localized groups of lymph nodes is common. This can occur without demonstrable involvement of the fascial planes, other than that which is present at the place where the glands are suppurating. In other words, it is then strictly localized, and in fact the suppuration brings about localization. In such cases incision should be delayed until fluctuation or inflammation of the skin is present, provided that there is no sepsis and no evidence of deep involvement. There is another type of case in which the individual glands on the deep fascia cannot be felt and there is no localized suppuration. There may be only a slight fullness along the sternomastoid muscle with well marked tenderness on pressure or manipulation of the muscle due to inflammation of the nodes beneath it which lie on the carotid sheath. In these, suppurative disintegration of the glands takes place late or not at all, and sepsis and blood culture examinations become the important indicators. Spontaneous recovery is possible and common, but an occasional case will tax the best surgical judgment. In two cases in which large superficial abscesses were evacuated the inflammation did not remain confined to the glands, though free drainage had been established, and there

took place extension into compartments of the deep fascia. This changed the picture and necessitated the introduction of drains into the fascial compartments invaded. In one of these, case No. 15, the infection lay dormant in a lymph node in contact with the submaxillary salivary gland for several months, when suddenly it became acutely inflamed, involving the salivary gland compartment, and spread to the pharyngomaxillary and parotid compartments. Before the inflammation was under control it had invaded the carotid sheath.

In the other case, No. 10, the infection began in the submaxillary and upper cervical lymph nodes, rapidly formed an abscess which was promptly incised and drained. Everything progressed as though it were going on to a prompt recovery in a few days, when suddenly the infection extended along the fascia, invading the parotid, submaxillary and pharyngomaxillary compartments, and the sheath of the vessels high up. It is to be concluded therefore that if the infection remains superficial, and confined to the lymphatic elements, simple drainage incision through the skin and superficial fascia into the abscess cavity will bring about prompt recovery. If, however, the deep fascia is involved extension is to be expected, and the compartments invaded must be drained. Adherent, inflamed lymph nodes on the jugular sheath make ligation and resection a slow and tedious procedure. In the case No. 12, from which the photomicrograph was made, showing polynuclear infiltration of the vein wall, there were adherent, enlarged and inflamed nodes on the sheath which were carefully dissected off. Under the microscope these also showed polynuclear infiltration but no visible abscess. It would seem entirely reasonable to conclude that infection of the vein wall may be caused by infected lymph nodes. In this type of case there should be no hesitation to divide the belly of the omohyoid muscle if it interferes with proper exposure.

PREVERTEBRAL FASCIA INFECTION.

This group constitutes the well-known retropharyngeal abscess as seen in infants and children. There were three cases in this series, the ages of which were 8 months, 9 months and 6 years. An abscess in this locality tends to extend downward, so that

when it reaches the hypopharynx the swelling, protruding forward, caps the larynx and seriously embarrasses respiration. If in addition it extends laterally it may endanger the pharyngomaxillary space high up, or the carotid sheath lower down. I have never seen lateral extension of a typical midline retropharyngeal abscess.

I have observed one very unusual case, not included in this series because it was never proven to be inflammation of the cervical fascia. This case, after considerable study with orthopedic surgeons and internists, was attributed to myositis, possibly hemorrhagic, involving the muscles attached to the cervical spine, occiput and mastoid, viz., the trapezius, recti capiti, obliqui capiti, splenius and complexus. The patient made a spontaneous recovery. My reason for mentioning the case is that infection of the prevertebral fascia had to be ruled out, and to call attention to the character of the torticollis. The muscles mentioned were splinted in contracture, causing the chin to be drawn toward the same side with elevation of the shoulder. This is the opposite of what takes place when the sternomastoid muscle is splinted in contracture by infection beneath it.

When a patient with the typical retropharyngeal abscess is seen before respiratory embarrassment has occurred, simple peroral incision is usually promptly effective. If not, Dean's[5] external route for drainage is an excellent and easy method of approach. Where there is already severe dyspnea from respiratory obstruction it may be necessary to first relieve this by tracheotomy, particularly if there is considerable exhaustion. It may not be possible to make an exact diagnosis before this is done. Years ago one such case fell to my lot. It is reasonable to suppose that if neglected, these otherwise simple cases of retropharyngeal abscess die of asphyxia or pneumonia following rupture into the air passages, rather than by extension into the chest.

BUCCOPHARYNGEAL, VISCERAL OR PRETRACHEAL FASCIA INFECTION.

This is one of the most important fascial layers in the neck, and is given emphasis because of its contained viscera. In this series there were two cases, one of which, case No. 4, was mentioned under etiology as having been caused by the trauma

induced by an unmasticated bread crust. Infections beginning in the hypopharynx which penetrate the visceral walls involve this layer. Swelling is apt to appear in the hyoid and laryngeal regions laterally. When the infection is high up, lateral to the naso- and oro-pharynx, this becomes a pharyngomaxillary space infection.

This is the type of case which may sometimes be drained by internal incision, working through the laryngeal or esophageal speculum. In doing this in the region of the pyriform sinus it is important to avoid cutting the internal branch of the superior laryngeal nerve. Dysphagia, dyspnea and hoarseness are to be expected. If internal drainage does not check its progress, external drainage should be done, as downward extension is a possibility. In the bread crust case there occurred an abrasion of the right wall of the hypopharynx near its junction with the esophagus. An abscess developed at this point in the wall accompanied by moderate diffuse swelling of the neck over the region lateral to the larynx. It was drained by peroral incision through a laryngeal speculum, and the patient made a complete, but slow, recovery. For a number of days an external incision was under consideration, but was not done because drainage was good and the patient was not septic nor dyspneic. This type of case should be closely watched for downward extension, for involvement of the vessel sheath and for laryngeal and tracheal obstruction.

There was one other case, No. 21, classified under infection of the visceral fascia. The patient had received much conflicting medical advice, which, coupled with a natural desire to avoid surgery, brought her to the operating room late. The swelling and tenderness began in the region of the thyroid gland, and being a negress, redness of the skin could not be easily seen, though fluctuation was present. A week or more elapsed before the patient could be convinced that there was pus in the neck. It is interesting to note that while there was great increase in the degree of neck swelling on both sides there was at no time downward extension dangerously close to the clavicle. When it was finally incised over the posterior border of the sternomastoid muscle all four fingers of the hand could be passed between the

esophagus and the vertebral column to the opposite side of the neck. A very large quantity of pus was evacuated. At no time was there dyspnea, hoarseness, aphonia or dysphagia. In other words, no visceral compression or functional embarrassment of the recurrent laryngeal nerves was present. After complete healing had taken place, and the patient discharged, the opposite side of the neck began to swell, this time being characterized by marked induration. Localization was slow, and when the lower part of the posterior triangle became involved incisions, one high and one low, were made, resulting in gradual recovery. Considerable cellulitis existed this time in contrast with the suppurative softening found previously on the other side. Downward extension into the chest would no doubt have been fatal. I am under the impression that this complication is rare except in cases of esophageal trauma and perforation.

SURGICAL APPROACHES FOR DRAINAGE.

The actual line of incision through the skin is of secondary importance, providing it is made long enough to give a wide exposure. Mosher, who first definitely outlined the approach to the deep fascial spaces of the neck, wisely advises a generous T-shaped incision which gives a wide, flat exposure. Through this, veins which are apt to flood the field with blood are more easily seen and tied. The cross-bar of the T runs parallel with the border of the jaw and close to it. Its object is to expose the submaxillary salivary gland. After the facial vein is tied and cut the lower border of the gland is elevated near its lateral extremity, and the finger inserted beneath it and carried backward and upward along the deep layer of its fascia until the stylomandibular ligament is felt beneath the angle of the jaw. It is then in position to enter any of the three uppermost deep fascial spaces. This is a very easily felt band of fascia, and after being located, the finger is carried upward along it until the styloid process is felt. By carrying the finger upward and external to the styloid process to the skull base it has entered the pharyngomaxillary space. By carrying the finger forward from the ligament along the inner side of the jaw it gets into relationship with the side of the tongue, floor of the mouth and

the region of the tonsil. One of the fingers of the other hand may be inserted into mouth and pharynx as a guide, being careful to observe aseptic technic by changing the glove when withdrawn. The vessel sheath lies deep to the styloid process, and is, therefore, also drained at that point by this maneuver.

If the infection is mostly deep and more or less limited to the pharyngomaxillary fossa the dissection exposing the submaxillary gland is comparatively easy because the tissues superficial to the gland are not inflamed and swollen. In most of my cases the submaxillary region was inflamed and considerably swollen so that it was more difficult to recognize structures. In these I have used a single long incision running parallel to the border of the jaw and extending nearly to the mastoid tip. The deep fascia is exposed, carefully snapping and tying all vessels. At a point directly behind the angle of the jaw a closed blunt artery clamp is thrust through the deep fascia, and the opening enlarged sufficiently to give room for the introduction of a finger. Passing the finger beneath the angle of the jaw the stylomandibular ligament is felt for, and by the maneuver above described enters the desired fascial space. If it is desired to expose the vessel sheath I prefer to work through a longitudinal incision having its upper end at or near the angle of the jaw and running along the anterior border of the sternomastoid muscle. At the upper extremity of this incision one may carry out the necessary finger maneuvers to enter the fascial spaces after locating the stylomandibular ligament. Inflamed, swollen and indurated tissues are concededly more difficult to recognize than those not thus involved, and a cadaver dissection is not always feasible nor desirable. With a few simple rules such as are here outlined this detailed recognition is not necessary. When, however, it is necessary to drain the vessel sheath or resect the jugular vein, anything short of complete exposure is unthinkable.

Dean[6] has recommended that all retropharyngeal abscesses be drained by the external route, incising along the anterior border of the sternomastoid muscle lateral to the larynx, reaching the visceral fascia, and following this around to the prevertebral fascia, thrusting the finger or a blunt instrument through into the abscess cavity. In cases where there is mechanical respiratory

obstruction this method would avoid preliminary tracheotomy. As he states, it can also be reached through an incision along the posterior border of the muscle, but the anterior incision minimizes manipulation of the carotid sheath and avoids the underlying sympathetic nerve.

The following is a brief group summary of the surgical approaches:

1. Prevertebral fascia infection or retropharyngeal abscess.
   (a) Direct incision of the pharyngeal wall through the mouth.
   (b) Incision along anterior border of sternomastoid muscle lateral to the larynx as recommended by Dean.
2. Pharyngomaxillary, parotid and submaxillary space infection.
   (a) Approach through submaxillary space after elevating the gland as recommended by Mosher.
   (b) Approach beneath angle of jaw directly without elevating the gland.
3. Carotid sheath and visceral fascia infection.
   Incision along anterior border of sternomastoid muscle as for jugular resection.
4. Posterior triangle infection.
   Incision along or behind the posterior border of sternomastoid muscle.

COMPLICATIONS.

When mentioned as they occurred in individual cases their multiplicity is shown, and also the combinations in which they appeared.

Rupture through the wall of the external auditory canal took place in one patient, case No. 7, in whom there was severe suppuration in the parotid sheath. Such a condition demands careful examination to differentiate it from an acute otitis media. Both conditions may be present at the same time.

Thrombosis of the internal jugular vein in one patient, case No. 24, was the direct result of extension from the submaxillary and parotid spaces, there having been at no time involvement of the pharyngomaxillary space. Because of the subsidence of the neck swelling and the development of abdominal distention it was the opinion of a number of consultants that the cause of the sepsis was in doubt, and exposure of the vascular sheath and resection

of the internal jugular vein was delayed twelve days. At this time the vascular sheath was exposed, and the vein found completely thrombosed and so much macerated that ligation or resection was impossible. Following this there occurred thrombophlebitis of the veins of both legs with subsequent abscess formations requiring drainage. There also occurred the same pathology in one side of the pelvis necessitating drainage. Blood cultures showed streptococcus hemolyticus. Cystitis and choked discs added to the patient's misery. At the time of writing this patient is still in the hospital, a sojourn of three months.

Thrombosis of the internal jugular vein and cavernous sinus occurred in one patient, case No. 8, and caused the only death in the series.

Acute mastoiditis with sigmoid sinus thrombosis occurred as a complication in one patient, case No. 16, and was followed later by the development of an abscess in the hip joint which required drainage. In this patient there was also homolateral paralysis of the tongue and of the pupillary fibers of the cervical sympathetic nerve. Blood culture showed streptococcus hemolyticus.

Phlebitis of the internal jugular vein without thrombosis was demonstrated in one patient, case No. 12. The father of this patient is a physician to whom the nature of the condition could be fully explained. On account of the persistence of the sepsis, and despite negative blood cultures and very slight neck swelling, consent for jugular resection was readily given. The presence of polynuclears in the vein wall and adherent lymph glands tells its own story and throws much light on these infections.

It would seem that thrombosis of the internal jugular vein may take place in at least two ways. First, from thrombophlebitis of its small tributaries high up in the pharyngomaxillary space, especially in the region of the tonsil. Waldapfel[4] is again referred to in this connection. Second, by extension of the inflammation from the sheath and from contacting lymph nodes. This was demonstrated by case No. 12, above referred to, where there was no thrombus formation, and is interpreted as being the early prethrombus stage.

Secondary hemorrhage from the small veins of the pharyngomaxillary space occurred in one patient, case No. 11, on the sec-

ond day after operation. Removal of the stiff rubber drainage tubes and substitution of iodoform gauze promptly checked the bleeding. Subsequently soft rubber cigarette drainage material was used and no more bleeding occurred. In this patient there also occurred paralysis of pharyngeal branches of the glossopharyngeal nerve, paralysis of laryngeal branches of the vagus and paralysis of the spinal accessory nerve.

The following is a list of complications which occurred in these cases:

1. Rupture into the external auditory canal.....Once
2. Thrombosis of the internal jugular vein.....Twice
3. Acute mastoiditis with thrombosis of the sigmoid sinus.....Once
4. Phlebitis of the internal jugular vein without thrombosis.....Once
5. Thrombosis of the internal jugular vein and cavernous sinus.....Once
6. Thrombosis of veins of both legs followed by abscess formation....Once
7. Thrombosis of veins in right side of pelvis followed by pelvic abscess.....Once
8. Abscess of the hip joint.....Once
9. Bilateral choked disc.....Once
10. Secondary hemorrhage from veins in pharyngomaxillary space.....Once
11. Cystitis.....Once
12. Paresis of the ninth, tenth and eleventh cranial nerves.....Once
13. Paresis of the twelfth cranial nerve.....Once
14. Paralysis of the pupillary fibers of the cervical sympathetic nerve..Once
15. Blood stream infection demonstrated by positive cultures.....Twice

Extension into the chest or axilla and rupture of the carotid artery I have not yet observed.

SUMMARY OF FINDINGS.

1. Adequate drainage can be obtained only through the external route. If internal drainage has been done, and does not bring about immediate subsidence of the infection, external incision must be made without delay.

2. Phlebitis of the internal jugular vein, with or without thrombosis, is often present. There were five cases, 20 per cent. One died unrecognized; the others received adequate external drainage and lived. Evidence that involvement of the vein may occur early was shown in one of the cases that recovered. In this one there was polynuclear infiltration of the wall of the vein, erosion of the intima, and minute deposits of fibrin on the endothelium, without thrombosis.

Cases — Age and Sex	Etiology — Portal of Entrance	Regional Pathology							Treatment Received	Result
		Pharyngo-Maxillary Space	Submaxillary and Parotid Fasciae	Lymph-adenitis	Pre-vertebral Fascia	Visceral — Bucco-pharyngeal — Pretracheal	Carotid Sheath	Complications		
No. 1 Jennie R. — 9 mo., f	Naso-pharyngitis — Infect'd aden'd				Retro-pharyngeal abscess			None	Per-oral incision	Recovery
No. 2 Rose A. — 8 mo., f	Infected tonsils and adenoid				Retro-pharyngeal abscess			None	Per-oral incision	Recovery
No. 3 Joseph H. — 6 yrs., m	Infected tonsils and adenoid				Retro-pharyngeal abscess			None	Per-oral incision	Recovery
No. 4 Mary F. — 42 yrs., f	Hypopharynx trauma					Bucco-pharyngeal abscess		None	Incision through laryngoscope	Recovery
No. 5 Oscar B. — 19 mo., m	Infected tonsil	Lateral pharyngeal abscess						None	Per-oral incision	Recovery
No. 6 Kath. R. — 20 yrs., f	Pharyngitis and infected tonsils	Pharyngo-maxillary cellulitis						None	Non-surgical treatment	Recovery
No. 7 Center H. — 14 mo., m	Infected tonsils and adenoid	Pharyngo-maxillary abscess	Submaxillary and parotid	Secondary				Rupture into ext. auditory canal	External drainage	Recovery
No. 8 Leonard P. — 16 yrs., m	Infected tonsil remnant	Pharyngo-maxillary abscess		Secondary			Sheath infection	Thrombosis of internal jug. vein and cavern's sinus	Per-oral incision	Death
No. 9 Gilbert K. — 3 yrs., m	Infected tonsils	Pharyngo-maxillary abscess		Secondary with abscess				Lymph glands abscess	External drainage	Recovery

No. 10 Jane M. — 5 yrs., f	Pharyngitis and infected tonsils	Pharyngo-maxillary abscess secondary	Submaxillary and parotid infection secondary	Cervical glands abscess primary				None	External drainage	Recovery
No. 11 Shaw C. — 17 yrs., m	Pharyngitis	Pharyngo-maxillary abscess						Secondary hæmorrhage Paresis of 9th, 10th and 11th cranial N's	External drainage	Recovery
No. 12 Sally H. — 4 yrs., f	Trauma from adenoid operation	Pharyngo-maxillary abscess		Glands on sheath infected			Infected	Phlebitis of internal jugular	Ext. drainage and resection of jugular vein	Recovery
No. 13 Dorothy S. — 19 yrs., f	Otitis media and petrous temporal bone infection	Pharyngo-maxillary abscess	Parotid fascia infection					None	External drainage	Recovery
No. 14 Rose F. — 32 yrs., f	Infection in zygomatic fossa following local anæsthesia injection	Pharyngo-maxillary abscess	Parotid and submax. fascia infection					None	External drainage	Recovery
No. 15 Edythe R. — 26 yrs., f	Submaxillary lymph node suppuration	Pharyngo-maxillary abscess secondary	Submaxillary space abscess — primary				Carotid sheath infection	None	External drainage	Recovery
No. 16 Alfred S. — 7 yrs., m	Rhino-pharyngitis	Pharyngo-maxillary abscess	Submaxillary space infection	Lymph adenitis secondary				Mastoiditis — Sigmoid sinus thrombosis — Hip j'nt abscess — Paresis of 12th nerve — Paresis of pupillary fibers of cervical sympath. nerve	Ext. neck dr. — Mastoid-ectomy — Drainage of sigmoid sinus — Resection of int. jug. vein — Hip j'nt. dr.	Recovery

Cases — Age and Sex	Etiology — Portal of Entrance	Regional Pathology						Complications	Treatment Received	Result
		Pharyngo-Maxillary Space	Submaxillary and Parotid Fasciae	Lymph-adenitis	Pre-vertebral Fascia	Visceral — Bucco-pharyngeal — Pretracheal	Carotid Sheath			
No. 17 Eliz. W. — 6 yrs., f	Pharyngitis			Cervical gland abscess				None	External drainage	Recovery
No. 18 Joseph C. — 10 mo., m	Infected tonsils and adenoid			Cervical gland abscess				None	External drainage	Recovery
No. 19 Sylvia F. — 14 yrs., f	Trauma from tonsil operation	Small localized abs. beneath tonsil fossa muscle bed						None	Spontaneous drainage	Recovery
No. 20 Edgar W. — 21 yrs., m	Dental infection		Submaxillary fossa infection — Parotid fascia infection					None	External drainage	Recovery
No. 21 Charity D. — 53 yrs., f	Thyroid gland fascia-Lymph node			Lymph-adenitis on visceral layer		Pretracheal or visceral fascia abscess		None	External drainage	Recovery
No. 22 Daniel M. — 30 yrs., m	Dental infection		Submax. and submental triangles Infection with infection of floor of mouth					None	External drainage	Recovery

No. 23 Rose L. — 7 yrs., f	Dental — — Alveolar abscess		Submax. and submental triangles — Infec'n floor of mouth				Carotid sheath infection	Thromb. of int. jugular vein — Thrombosis of veins of legs, abscess formation — Thrombosis of pelvic veins followed by pelvic abscess — Bilateral choked disc	External drainage of carotid sheath Leg abscess drainage bilateral — Pelvic abscess drainage.	Recovery
No. 24 Louise B. — 47 yrs., f	Pharyngitis		Submaxillary and parotid fascia, and carotid sheath	Secondary — Without suppuration						Recovery

3. Swelling of the lateral pharyngeal wall is an early and constant sign. Look for it, but do not always expect to see increased redness. It may look pale.

4. Trismus is one of the most important early signs of infection in the pharyngomaxillary space. It is due to splinting of the internal pterygoid muscle by the advancing inflammation. It is explained by the applied anatomy. The constrictor muscles of the pharynx form the inner wall of the space, and the internal pterygoid muscle a part of the outer wall. The result of infection between the two is obvious.

5. Chills, sweats and high excursions of temperature are common symptoms and do not necessarily require jugular resection unless they continue some days after adequate drainage has been established. They do demand immediate external drainage of the deep fascial compartments.

6. Quinsy sore throat is the diagnosis most frequently made when they come referred. When seen early the differentiation must be made, and it is sometimes difficult. It is possible for a peritonsillar abscess to antedate the infection in the pharyngomaxillary space though I have seen none. If so, one might consider doing tonsillectomy.

7. The degree of external swelling may be small or barely distinguishable, so that by itself it is no criterion for estimating the necessity for external drainage. Absence of external swelling does not contraindicate external drainage if there is sepsis. Infection of a fascial plane may be present without visible evidence of pus. The microscope has demonstrated marked evidence of infection in just such cases. I am very much indebted to Dr. Harris P. Mosher for a slide which proves this statement.

8. Blood may remain negative despite the existence of a phlebitis, even when the blood to be cultured is withdrawn from the internal jugular vein.

9. Cervical gland abscesses usually remain superficial and localized, but occasionally the infection will spread along the layers of the deep fascia, with resulting change of picture requiring drainage of the compartments invaded.

10. Prevertebral fascia infection constitutes the well known condition called retropharyngeal abscess. When seen early and

drained before there is obstruction to breathing, recovery is prompt. If untreated, these cases die from asphyxia due to capping of the larynx by the protruding posterior pharyngeal wall rather than from extension of the infection into the chest.

11. Torticollis from infection under the sternomastoid muscle is toward the opposite side. Torticollis from infection along the paravertebral muscles and trapezius is toward the same side.

12. The ears should be frequently inspected, as there may be a coincident tympanomastoid inflammation. In this series there were two such instances, one of which went on to obstructive sigmoid sinus thrombosis.

13. The sepsis or septicopyemia may go on to a fatal termination without apparent increase in the severity of the local manifestations. This happened in my first case, the only death in the group. The cause of death was thrombosis of the internal jugular vein and cavernous sinus. The reason, I believe, was failure to drain externally because two consultants, a general surgeon and a laryngologist, advised against it. Thrombosis of the internal jugular vein was not suspected until the signs of cavernous sinus thrombosis appeared.

14. Chills are to be expected. Blood for culture should be taken during or immediately after the first chill. Blood cultures may become negative after the vein becomes corked by the thrombus.

15. Metastatic abscesses following thromboses in distant veins are common after thrombosis of the jugular has occurred.

CONCLUSIONS.

1. Cases of jugular thrombosis from infection in the planes of the cervical fascia are going unrecognized and dying from septicemia.

2. Absence of frank local manifestations of the presence of the infection, notably swelling, is common.

3. Delayed surgery is apt to be disastrous and engenders regret.

I wish to acknowledge my indebtedness to Doctors P. I. McIlroy and H. vonWedel of the New Rochelle Hospital Laboratory for their skilled interpretation of the microscopic pathology in connection with the above mentioned cases.

421 HUGUENOT ST.

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6. Ibid.

NOTES ON THE ILLUSTRATIONS.

Figure No. 1 shows the pharyngomaxillary fossa as seen from behind. The superior constrictor of the pharynx forms its inner wall, the internal pterygoid muscle and retromandibular process of the parotid gland its outer wall. The structures attached to the styloid process cross it, dividing it unequally.

Figure No. 2 shows the close relationship of the parotid and the submaxillary glands to each other and to the great vessels.

Figure No. 3 shows the point at and under the angle of the jaw where the layer of fascia on the deep surface of the two salivary glands thickens to form the stylomandibular ligament. This is the landmark which is felt with the finger in the approach to all three of these spaces.

Figure No. 4 shows the manner in which the internal pterygoid forms the outer wall of the space. It also shows the stylomandibular ligament.

Figure No. 5 is a diagrammatic cross-section drawing of the pharyngomaxillary space and the structures bounding it on two sides. It explains three important signs of infection in this space: swelling of the lateral pharyngeal wall by displacing the superior constrictor into the pharynx, trismus due to splinting of the internal pterygoid, and swelling of the parotid gland region by extension of the inflammation into the sheath of the gland.

Figure No. 6 shows a skeletonized drawing of the buccopharyngeal and visceral fascia, and explains how an infection may extend from the skull base to the chest. It also shows how an infection may encircle the trachea and esophagus.

Figure No. 7 is a skeletonized diagrammatic drawing showing the location of some of the fascial compartments in the neck, and their relationships.

Figure No. 8 is the same with the head omitted.

Figure No. 9 is the same, somewhat altered, but showing the same relationships.

Figure No. 10 is the same, altered to represent a trifolium.

Figure No. 11 shows the bony and muscular framework upon which the prevertebral fascia is reflected. When thus viewed from the front it has a similarity to the appearance of the inside of a tent when viewed through its open door. It explains why a true midline infection tends to remain so.

Figue No. 11-A shows a normal internal jugular vein from a newborn infant which died of congenital heart disease.

Figure No. 12 shows a section through internal jugular vein, case No. 12. *A*, Polynuclear leucocytes. *B*, Erosion of endothelium. High power.

Figure No. 13—Another field from the same slide.

Figure No. 14, the same under oil immersion.

Figure No. 15 shows a section through lymph node dissected off the jugular sheath, case No. 12, showing (*a*) polynuclear leucocytes near the capsule.

Figure No. 16 shows a cross-section through the whole of one side of a neck showing very extensive inflammatory involvement of the fascial planes and jugular vein which was thrombosed. All this without visible external swelling of the neck. Symptoms in this case were predominantly respiratory in character and the patient died after tracheotomy. It is obvious that there was no obstructive swelling of the tracheal membrane. For the use of this slide I am greatly indebted to Dr. Harris P. Mosher, whose case this was, and I also wish to thank Dr. D. K. Judd of the Massachusetts Eye and Ear Interne Staff for the lantern slide and photograph.

Figure No. 17 shows the same section under higher power, showing the inflammatory infiltration in and around the internal jugular vein.

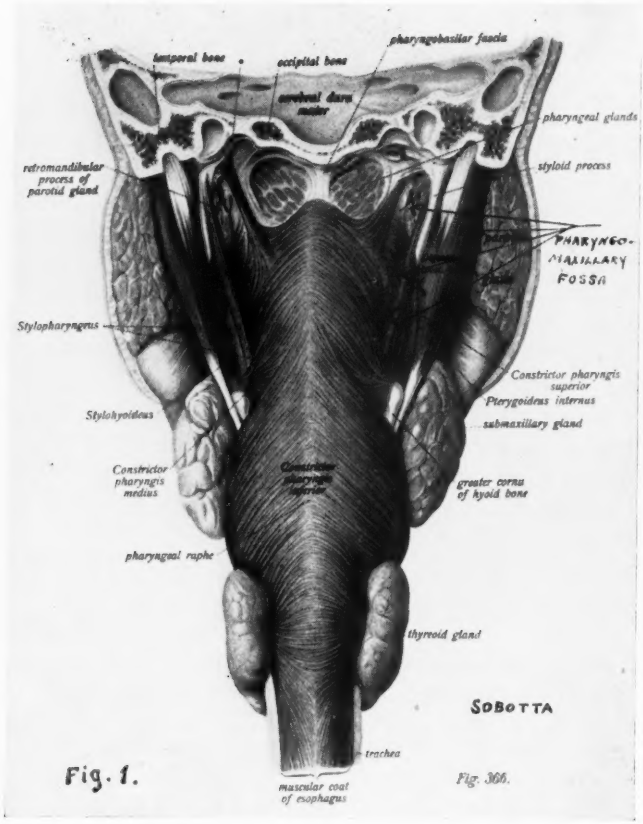

Fig. 1.—Pharyngomaxillary fossa from behind.

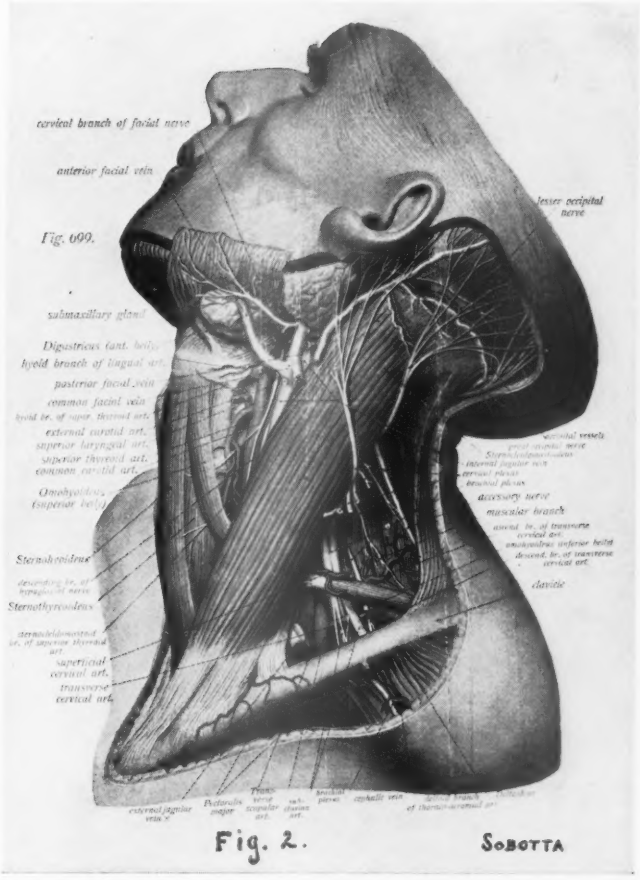

Fig. 2.—Close relationship of the parotid and submaxillary glands to each other and to the carotid sheath.

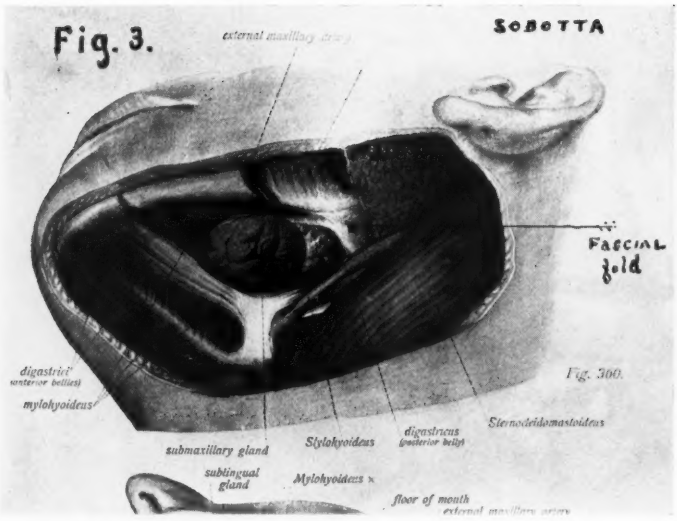

Fig. 3.—Fold of fascia between the parotid and submaxillary glands.

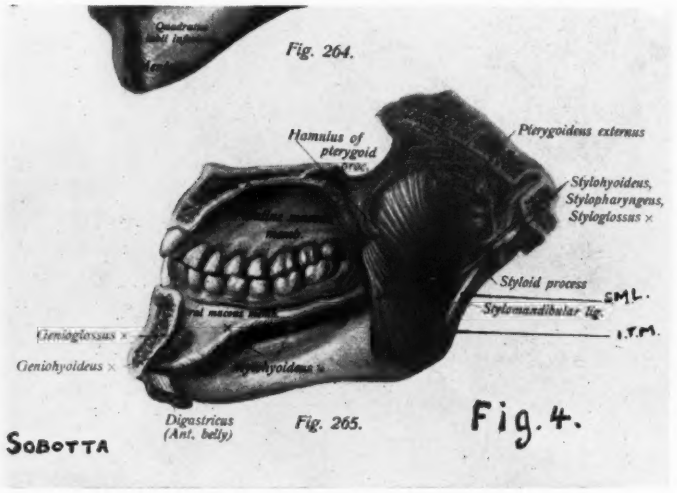

Fig. 4.—Stylomandibular ligament and internal pterygoid muscle.

Fig. 5.

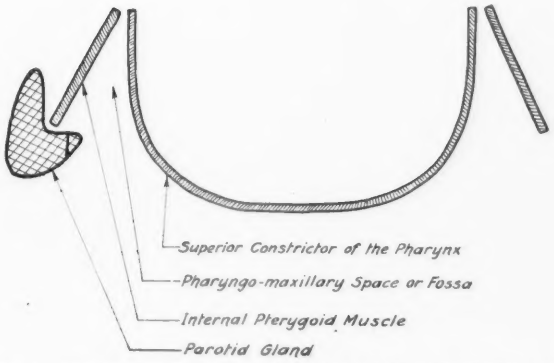

Fig. 5.—Diagrammatic cross-section drawing of the pharyngomaxillary fossa.

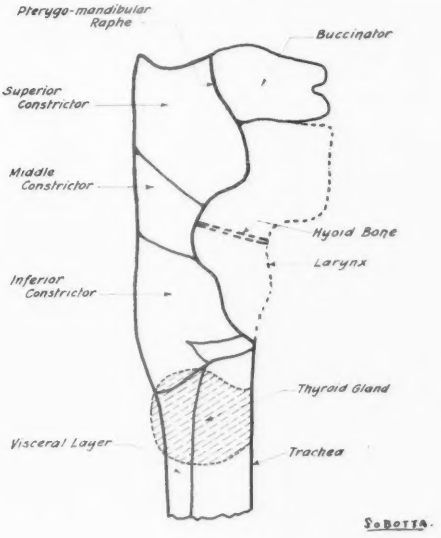

Fig. 6.

Fig. 6.—Skeletonized drawing of the buccopharyngeal and visceral fascia.

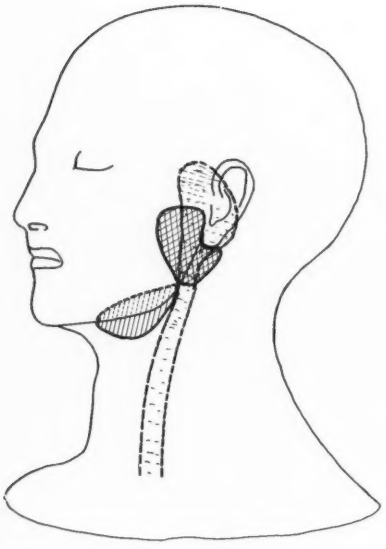

Fig. 7.

Fig. 7.—Shows location of fascial compartments in upper part of neck.

Fig. 8.

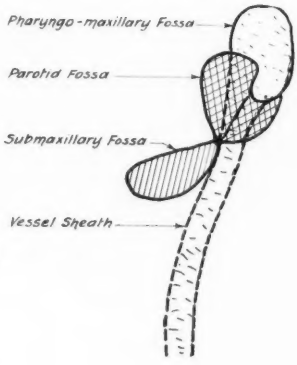

Fig. 9.

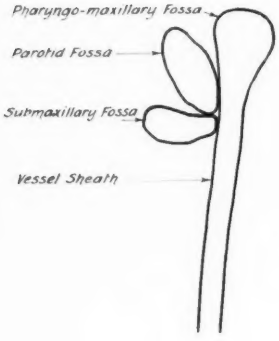

Fig. 10.

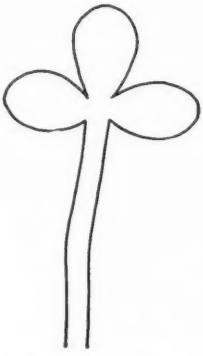

Fig. 10.—Trifolium.

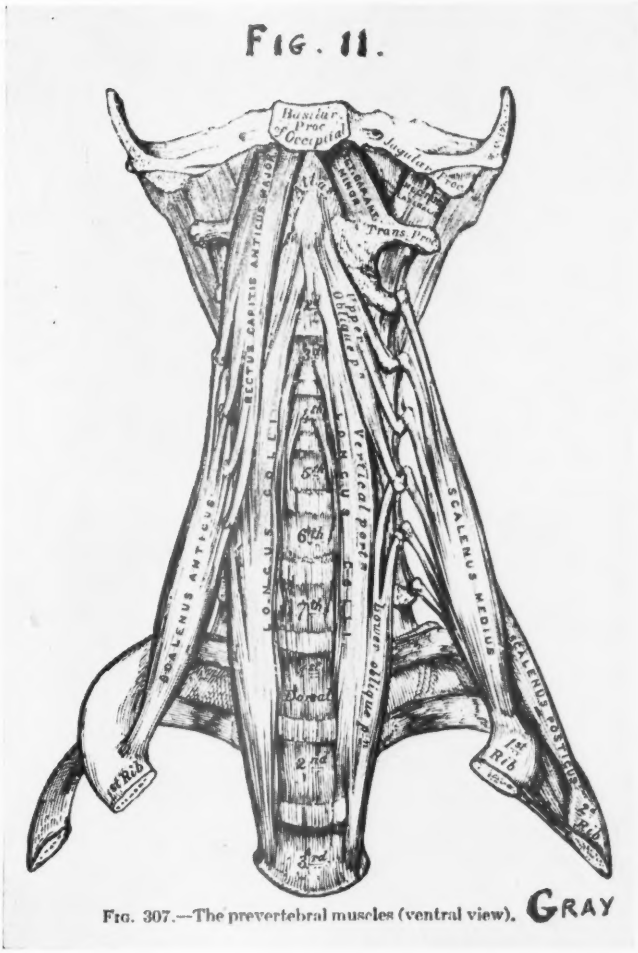

Fig. 11.—Shows the bony and muscular framework upon which the prevertebral fascia is reflected. When thus viewed from the front it has a similarity to the appearance of the inside of a tent viewed through its open door.

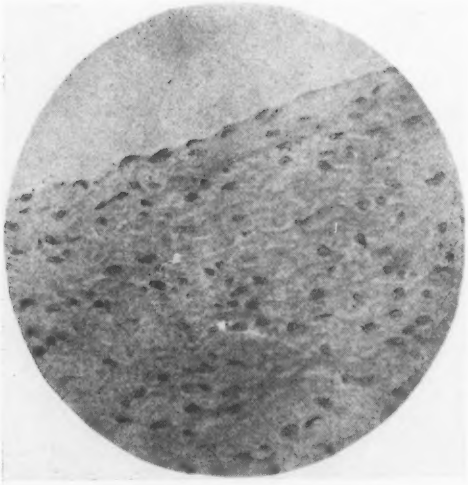

Fig. 11-A.—Normal internal jugular vein.

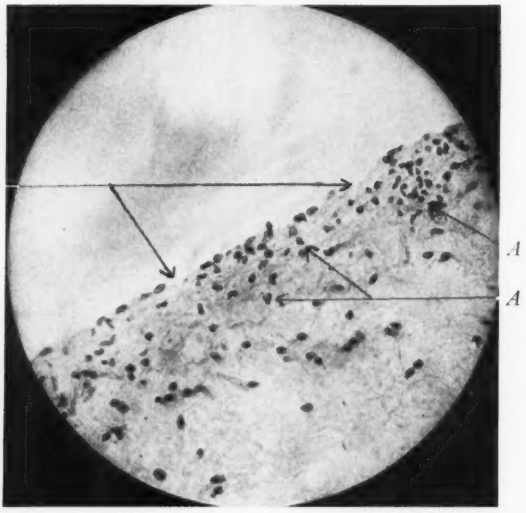

Fig. 12.—Section through internal jugular vein, Case. No. 12. *A*, Polynuclear leucocytes. *B*, Erosion of endothelium. High power.

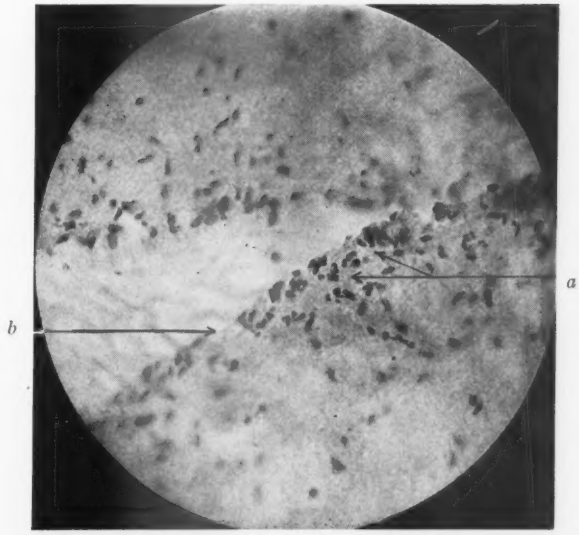

Fig. 13.—Section through internal jugular vein, Case No. 12. (*a*) Polynuclear leucocytes. (*b*) Erosion of endothelium. High power.

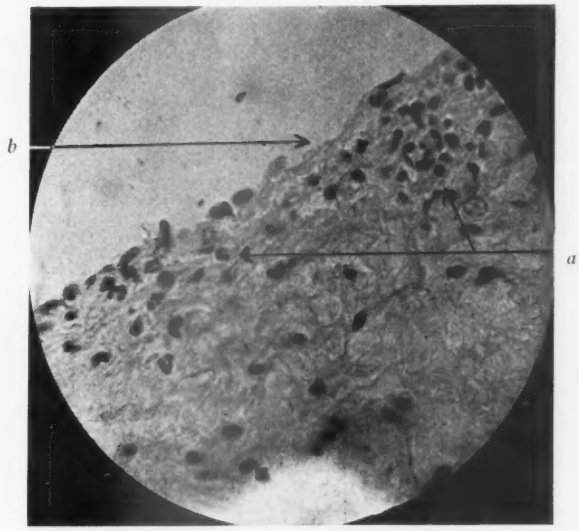

Fig. 14.—Same as Figure 12, with oil immersion.

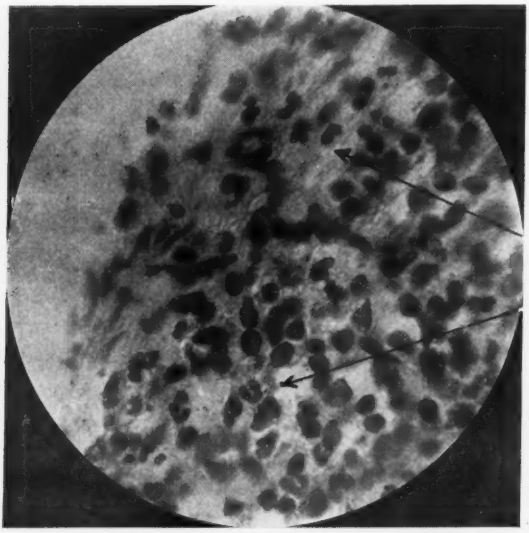

Fig. 15.—Section through lymph node dissected off the jugular sheath. Case No. 12, showing (*a*) polynuclear leucocytes near the capsule.

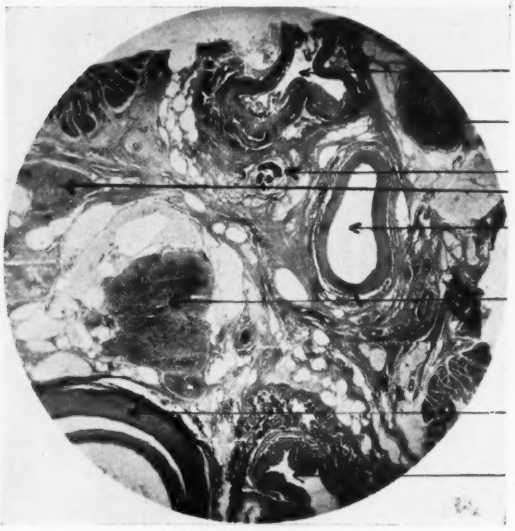

Fig. 16.—Cross-section through the whole of one side of the neck showing very extensive inflammatory involvement of the fascial planes and jugular vein. All this without visible external swelling of the neck. For the use of this slide I am greatly indebted to Dr. Harris P. Mosher, whose case this was. (Low power.) (*a*) Internal jugular vein. (*b*) Vagus nerve. (*c*) Common carotid artery. (*d*) Thymus gland. (*e*) Trachea. (*f*) Esophagus. (*g*) Lymph gland. (*h*) Lymph gland.

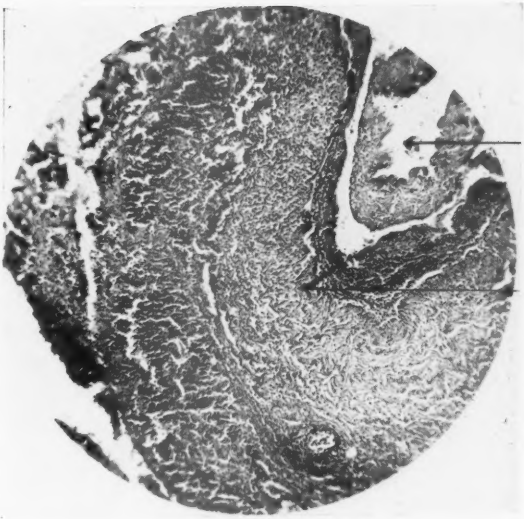

Fig. 17.—Same section under high power showing the inflammatory infiltration in and around the internal jugular vein. (*a*) Internal jugular vein. (*b*) Perivascular sheath.

LVII.

STUDIES OF NASAL CILIA IN THE LIVING MAMMAL.*

ARTHUR W. PROETZ, M. D.,

ST. LOUIS.

It is agreed that the behavior of cilia in the nose and sinuses is of sufficient importance in the scheme of defense against common colds and other initial infections to warrant the minutest study. It is obvious also, that information, in order to be of any practical value, must be derived from membranes as nearly like those of the human nose as possible, operating under conditions as nearly normal as they can be made. While, experimentally, the use of ciliated epithelium from amphibians and other lower forms is often expedient and sometimes unavoidable, deductions made from it and applied to human membranes are not tenable. Air breathing animals, as has been pointed out, are put to the necessity of supplying their own fluids to keep cilia beating, while the water inhabitants are not. The whole mechanism of air currents and the protection against them, constitutes one of the major factors in the resistance against infection. Furthermore, the cilia in the lower forms are frequently the means of locomotion, of food propulsion, or some other function wholly foreign to anything required of the human nose. We must insist, therefore, upon mammalian membranes.

It has also been reasonably pointed out[1] that extirpated membranes may render a false picture of ciliary function; that in their natural environment cilia are often quiescent but that they are stirred to activity by a slight stimulus. It is conceivable, moreover, that their mere extirpation may supply that stimulus; and that in the absence of circulation, nerve connections, body heat and humidity almost any abnormality of motion may occur.

*From the Department of Otolaryngology, Oscar Johnson Institute, Washington University, with the aid of funds granted by the Chemical Foundation.

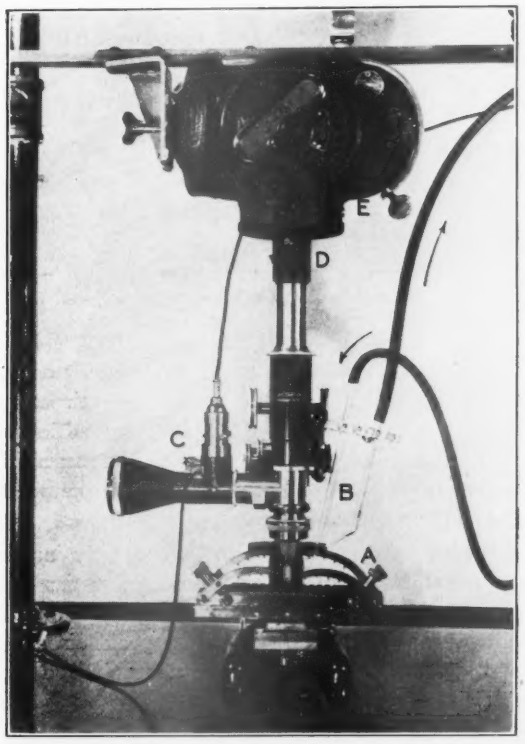

Fig. 1. Apparatus for examining and making motion pictures of nasal cilia in the living mammal.

A—Movable stage with clamp for holding head.

B—Capillary tubes for supplying and withdrawing fluids.

C—Opaque illuminator.

D—Reflex focusser.

E—Motion picture camera.

Thus far the microscopic examination of the human sinus lining in the living subject has not been feasible—at least not with the magnification necessary to reveal cilia. Although such an examination does not lie without the realm of possibility, the difficulties presented are enormous as will be seen from the description of the rabbit experiments which follows.

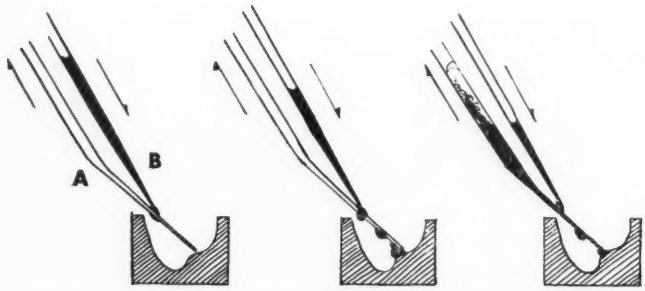

Fig. 2. Arrangement of capillary tubes for applying and removing fluids. *A*—Vacuum tube. *B*—Supply tube.

Lacking this final direct observation, it appears most practical and reliable to combine the parallel studies of extirpated human membranes, and the membranes of rabbits observed in the living animals, both under direct vertical vision with a magnification of 104 diameters, and a motion picture record to permit repeated examination and deliberate analysis.

As microscopy by transmitted light is obviously impossible in the living animal, it was necessary to devise a means of vertical opaque examination which would yield sufficient illumination for micrography at 1/30 of a second, and would also permit the supply *and removal* of fluids, and, when required, a stream of air for artificial drying.

A short description of the apparatus is necessary here to elucidate the observations and photomicrographs which follow.

The stage, condenser and substage mirror of the microscope are removed. The condenser carrier with its elevating and lowering rack and pinion is retained, and into this is fitted a clamp for fixing the rabbit's head in position. A Leitz "Ultropak" vertical illuminator with a 6.5 objective is employed, which makes available at once incandescent lamp illumination for routine study and arc light for photography. A 16X ocular and a firmly supported, quickly adjusted motion picture camera, with reflex focusser, complete the optical equipment. (Fig. 1.)

Vital to the success of the experiment is a pair of glass tubes attached to the frame of the microscope through a universal joint. These tubes are drawn to capillary tips and are so arranged that fluids may be supplied to and withdrawn from the microscopic field under observation at will—in fact, so efficiently that the membrane is never flooded with any excess fluid.

These tubes are used to study the effects of heat, cold, drying or the various treatment solutions upon the membranes. (Fig. 2.) Tube *A* is connected to a vacuum pump, constantly in operation. Its tip often appears in the field. Tube *B* is fastened with its tip in contact with Tube *A* about an inch from the tip of *A*. If fluid is dropped into Tube *B*, it escapes a drop at a time, the drops sliding down *A*, falling upon the membrane but being picked up by *A* before they can escape to flood the sinus or to embarrass the animal's breathing. This arrangement is easier to adjust than attempting to bring both the supply tube and the vacuum tube into the field at once.

The desired area of membrane is first brought into focus. The specimen is then lowered an inch or so, the tip of Tube *A* is brought into the field and the objective adjusted to bring it into approximate focus. The rabbit is now raised until the membrane is in focus once more, the tube all but touching it.

In preparation, a rabbit is anesthetized with an intravenous injection of sodium amytal to the point of complete analgesia. (The amount varies, usually about 65 mg. per kilo of body weight.) The clamp is removed from the microscope, and the animal's head securely fastened in it. A midline incision follows the bridge of the nose through its entire length, and the periosteum is elevated. Sutures through each side tied to the screws of the clamp serve as retractors. A chisel removes the entire bone, covering one nasal chamber, without perforating the nasal mucosa. This is not at all difficult. All bleeding must now be stopped by means of pressure, and the field cleaned with the aid of suction. When this has been accomplished, the membrane lying above the part to be studied is removed with a cataract or a paracentesis knife, the small flow of blood being taken up with the suction tip. The opposite side of the nose is undisturbed and is to be examined as a control at the end of the experiment.

The clamp is now attached to the microscope. What ensues is the most difficult stage of the procedure, namely the search for an area, not only covered with cilia, but of such a topography that it will reflect "high lights" from the condenser into the objective lens. It happens occasionally that a whole afternoon is spent in a fruitless search, or that a new animal must be resorted to. This is an additional reason for the motion picture record of the view, when it is finally satisfactory.

Concurrently with these experiments every available piece of human nasal mucosa removed at operation by the members of the staff is examined in like manner and under conditions of tem-

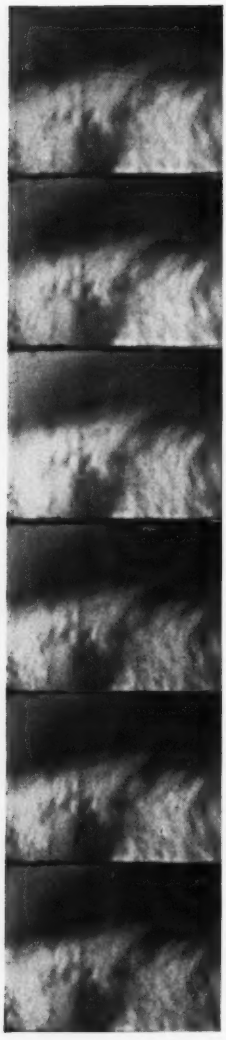

Fig. 3. Normal nasal mucosa in a living rabbit. (As reproduced, enlarged 416X.)

perature and humidity as nearly like those of the nose as is feasible. Thus far, no gross differences have been recognized, but it is not intended even to suggest that they do not exist, until a very large amount of material shall have been seen, and until the errors possible to the rabbit experiment have been reduced as far as is humanly possible.

Clinically we are concerned less with such problems as the nature of the ciliary impulses and their mechanism of coordination, than with those conditions which permit, pervert or destroy their maximum efficiency; and the present experiments are directed specifically to the latter.

The mucus which coats the membrane sometimes renders it difficult to see the ciliary motion. This may be brought out either by blowing upon it, thus slightly drying it, or by diluting it with Ringer's solution, or by a combination of the two. This, it is admitted, does not constitute an examination of the mucosa absolutely in its native state, but it is the nearest approach available at present, and ciliary action thus observed does not appear to differ in any respect from that in the untouched areas.

The rapidity of motion is somewhat variable, and depends at least partly upon the temperature, the moisture and, in the extirpated specimens, upon the time elapsed since removal. The removed specimens on the whole survived temperature variations below normal much better than relatively slight deviations on the

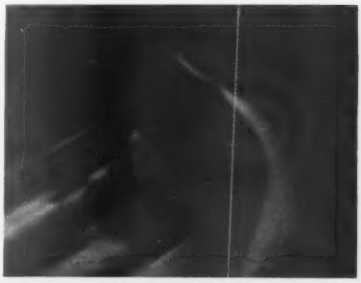

Fig. 4. Tip of capillary tube seen in the field, withdrawing fluid from the surface.

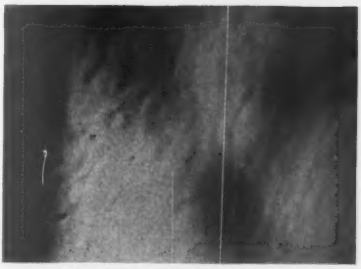

Fig. 5. Short wave forms. These are scarcely waves at all, but irregularities produced by the pulsations of bunches of cilia. The individual dimples do not travel along the surface but appear and disappear with the pulsations of the cilia.

Fig. 6. Longer wave fronts than Fig. 5 but observed in another part of the same specimen.

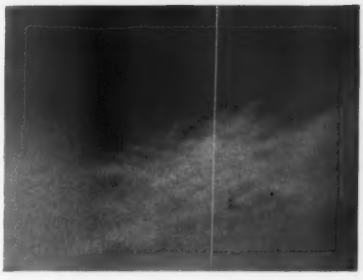

Fig. 7. Ciliary pulsations seen through the blanket of mucus.

These illustrations are reproduced from 16 mm. Ciné film. The magnification here is 636 diameters.

Fig. 8. Human maxillary mucosa, showing ciliary activity at points where the light is reflected. The mucus in the shaded areas obscures it. (Magnification 480X.)

Fig. 9. Portion of the above, showing the form of the ciliary propulsion. (Magnification 636X.)

Fig. 10. Human mucosa thinly submerged in Ringer's solution. Slight ciliary movement seen, right and left. (318 X.)

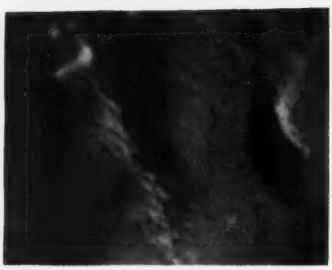

Fig. 11. Central portion of Fig. 10 (636 X). 30 seconds later. Below at left center activity appears as the solution is withdrawn.

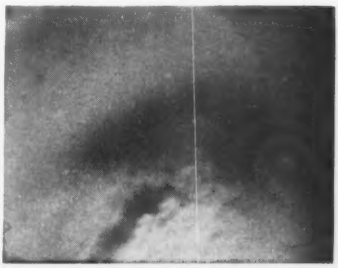

Fig. 12. Showing the action of drying. Rabbit mucosa (636 X). The area in the foreground has been blown upon, through a capillary tube.

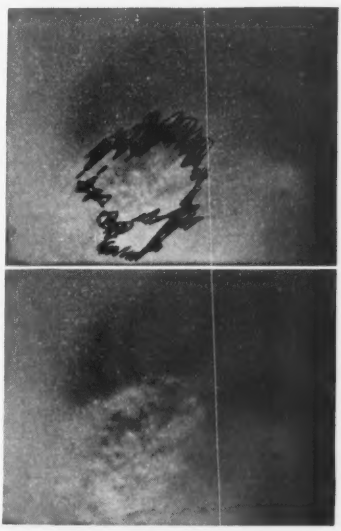

Fig. 12. The drying continues. At this point activity has ceased in the central area. A fringe of ciliary activity is present which widens as the fluids recede. In the margins of the photograph activity can be made out only by the movement of the dark dots which are minute bubbles in the mucus coat.

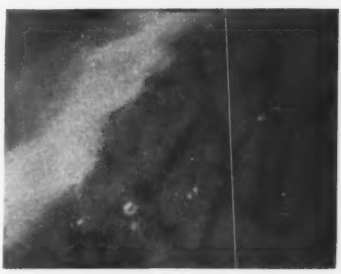

Fig. 13. Showing ciliary activity on the left, capillaries on the right. The bubbles cross this field in about 3½ seconds. (Magnification 636X.)

fever side. It has not been possible in these experiments to verify Wharton's observation of the retarding effect of light upon the cilia[3]. It was suspected that a slowing action of some kind was at work in the early experiments, which, since it disappeared on shutting off the light, corresponded fairly accurately to Wharton's description. However, the introduction of a cooling chamber between the source of light and the specimen terminated the phenomenon, which was therefore attributed to heat. Wharton in his communication describes his source of light as a 110 v. Pointolite, but does not mention a cooling chamber.

It may be said, in passing, that upon twenty-two specimens removed at operation, some of them from sinuses with the worst imaginable chronic infections, the cilia were beating vigorously, and to all appearances normally in every case, with a single exception. This series will be reported elsewhere in detail.

The time required for a single cycle of motion in both rabbit and man, varied from one-fourth to one-tenth second. This was estimated by counting the film frames of slow-motion pictures of known speed.

The wave forms seen from above are usually of the rounded and circumscribed, or "dimple" variety previously described, although occasionally the length of the wave front may be many times the distance between crests. Ordinarily the appearance is that of minute bunches of cilia, each beating rhythmically and not always exactly at the same speed as its neighbors. Sometimes the energy of a given area appears to flag. Mechanical irritation apparently is less effective in reviving it than moistening it with fresh Ringer's solution. Strips of membrane have been kept active upon the microscope stage for six hours and longer, without any further precaution than an occasional moistening with the solution.

Looking down upon the membrane confirms a previous suspicion that the direction of wave propagation is, or at least can be, in the opposite direction to that of the effective stroke. In a preliminary note[2] this was recorded, but there was then some doubt whether or not the stroboscopic effect of one row of cilia viewed through another in profile might not account for the phe-

nomenon. Since that time, however, the mucus coat has repeatedly been seen, from above, to slip in the direction opposite to the wave propagation. Viewed from this angle, the optical illusion could not occur.

Thus far, the most important observation, from a clinical standpoint, is the effect of drying. This is brought about sometimes by blowing upon the entire specimen, at others by projecting a jet of dry air through one of the capillary tubes. These experiments were done in the summer with the room temperature closely approximating body temperature and any cooling effect was confined practically to that of evaporation.

A small area under observation was dried and the cilia in this area ceased beating. A fringe of activity persisted so long as sufficient moisture remained. This circle of activity enlarged as the dry spot grew and a wider area began to dry. If this was allowed to continue for fifteen to eighteen minutes, the action could not be re-established by moistening. Flushing with physiologic sodium chloride solution or Ringer's solution, after a shorter time, however, restored the cilia and the same cycle recurred. At first, after flushing, only the brilliant reflecting surface of the fluid could be seen. Presently a small area of activity appeared through the surface of the evaporating fluid. Then as evaporation continued, the dry spot recurred at the center of the area, with the fringe of cilia as before.

I am convinced that this approximates a condition which is produced in the nasopharynx by jets of inspired air projected there through the agency of anatomic deviations and abnormalities. I believe also that a partial closure of the nasal chambers, brought about by chilling, local irritation, intestinal upsets or any other of the preliminary stages of an acute cold, produces this condition and, by interfering with proper ciliary propulsion of the mucus, lays the membrane open to infection. This conviction, reported in a communication to the American Medical Association at its last meeting, gains weight in view of the present microscopic findings. These seem to me to indicate to clinicians the extreme importance of maintaining equal distribution of nasal respiration, especially in the early stages of acute infections, and

of preventing stasis with its attendant irritation and resulting break in the protective barrier.

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LVIII.

FRACTURE OF THE STYLOID PROCESS AND ITS TONSIL FOSSA COMPLICATIONS, WITH REPORT OF A CASE.*

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PHILADELPHIA.

Spontaneous fracture of the styloid process of the temporal bone, perhaps better termed fracture without obvious relation to traumatism, is apparently of rare occurrence. Without doubt many cases have not been reported, and distorted position of the process in the tonsil fossa may have represented unrecognized fracture. This could readily happen with the difficulty in adequate X-ray demonstration. In the special case cited below, the plate of an examination made almost immediately after removal of the distal portion showed the fragment apparently intact in the original position. Careful subsequent X-ray studies proved the first to have been due to an exact alignment of the opposite styloid process, obscuring the picture on the operated side. Fractures coincident with some crushing injury to the skull are relatively frequent but do not belong in this category.

From a survey of the available literature, barely a half dozen cases, including the one here reported, appear to be chronicled as true spontaneous, or nontraumatic fractures, and the etiology is ascribed in three of them to a massive effort of swallowing or severe fit of coughing. The complex arrangement of muscles comprising the hyoid apparatus, with the hyoid bone acting as a fixed base, and the binding down of adjacent fascia, would afford, in unusual or complicated movements, and especially in strained position, an opportunity for displacement of the process. The possibility of pathologic fracture in these cases must not be overlooked. Deficiency in calcium content and delayed union of the two ossification segments should likewise be important. The first

*Presented before the Annual Meeting of the American Laryngological Association, Washington, D. C., May 9, 1933.

Fig. 1. Fracture of styloid process. Case reported by Dr. Pfahler.

case was reported by Pfahler[1] of Philadelphia in 1911, under the caption "Technique for the Isolated Roentgenogram of the Processus Styloideus." Though reported in a German journal, the patient consulted La Place in the Medico-Chirurgical Hospital, Philadelphia, in 1908, for examination of a tumor-like induration in the tonsillar region, which interfered with singing and caused painful swallowing. The roentgenogram showed fracture of the styloid process, with the proximal portion of the distal fragment drawn backward into the tonsillar area. There was an unusually long process on the opposite side. A difficult tooth extraction under gas six months before may have had some etiologic bearing in this case.

Goris,[2] in Belgium, reported two cases in 1900, one occurring suddenly in an effort of swallowing during which the patient was taken with a fit of laughter, and the second during a violent fit of coughing. Swallowing in each case aggravated the pain, and diagnosis was confirmed by digital examination.

Karl Specht,[3] in Germany, reported a similar case, also occurring during mastication. In his somewhat extended monograph, which included studies in the anatomy, physiology and development of the hyoid apparatus and discussion of the fracture mechanism, he states that his search of the literature revealed but two

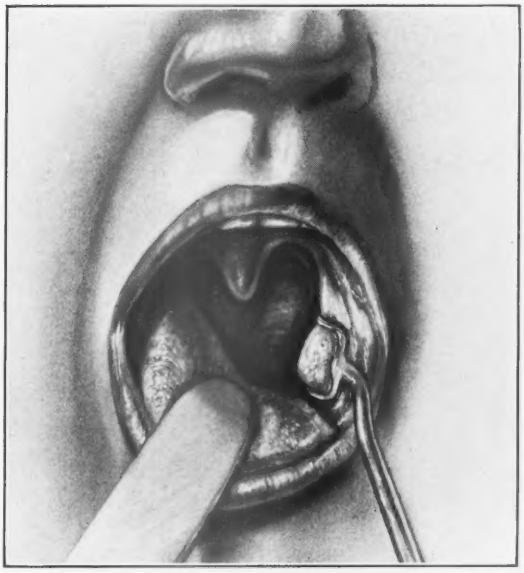

Fig. 2. Protrusion of styloid fragment in the tonsil fossa.

similar cases, those above credited to Goris. We are indebted to Harburger,[4] in Paris, for the most extensive study which has been published. In the fourth section of his paper, that on the normal and abnormal styloid, he presents forty-nine references, including eight of his own, in which the styloid-hyoid apparatus was studied. In one of these a spontaneous type of fracture is suggested and ascribed to external pressure.

The final case of the group is the subject of the special case report concluding this paper. A radical mastoid operation performed thirteen years before and the roentgenographic evidence of softened adjacent bone are the only known contributing factors. The only injury the patient could recall which might have any traumatic relation occurred about twelve years ago. While riding in an automobile her head was bumped against the top of the car. This occurred so long ago that it presents little significance in the recent situation.

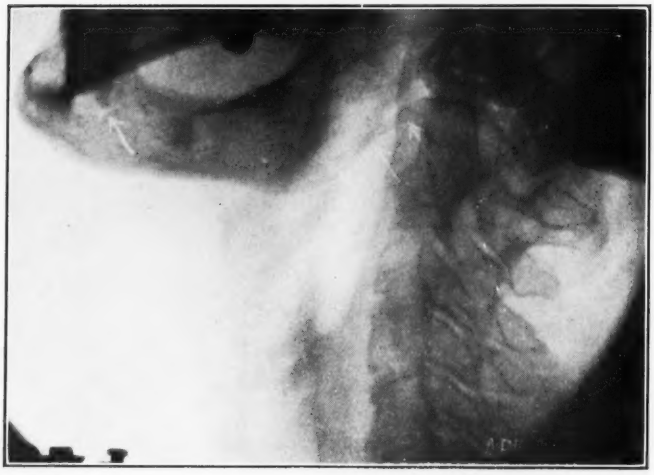

Fig. 3. Fracture of the left styloid process. Proximal end dropped downward.

In addition to this group, Patterson[5] suggests a possible fracture of the process during an epileptic attack in one of his patients, but it was not fully demonstrated.

Many references have been made to styloid disturbance in the tonsil and adjacent pharyngeal region due to elongation and malposition of the process, both before and after tonsil removal, with dysphagia the predominant symptom. In the search for relief, suspicious middle turbinates have been removed and adjacent glands scrutinized and operated upon.

In a study of the etiologic background, the same group of factors would seem to apply to fracture and malposition of the process without fracture. The principal ones are suggested: (1) The complexity of muscular and fascial pull in the anatomic make-up of the hyoidean apparatus; (2) imperfect ossification in the styloid process itself; (3) unrecognized traumatism, both intrinsic and extrinsic. Any combination of these factors may be present in producing the discomfort and malposition which suggest the diagnosis. The hyoid bone represents a fairly fixed base.

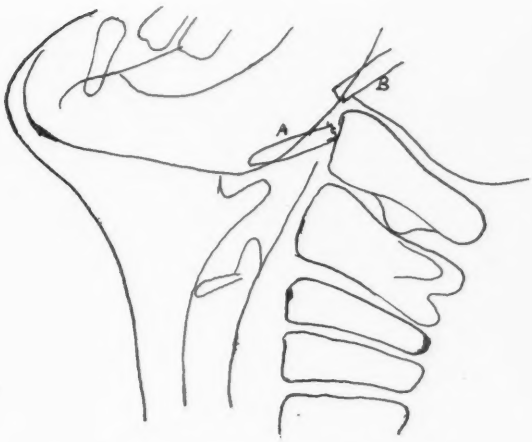

Fig. 4. Diagram illustrating positions in Fig. 3.

The supra-hyoid muscles connecting with the mandible, tongue and cranium, the middle constrictor of the pharynx and in an associated degree the infrahyoid group link with the stylohyoid and styloglossus in a most complex muscular action—such as occurs in forceful swallowing, choking or severe coughing. If these be inadvertently combined or interrupted by laughter, sneezing or other surprise movement, and especially in the presence of an elongated or maldeveloped process, the stage for displacement or fracture is complete.

Much has been published in this and other countries on the throat complications, particularly from an elongation of the styloid process. According to Kulvin,[6] Marchetis first refers to the ossification of the stylohyoid ligament in 1652, and Wemlicher of Vienna, in 1872, reported the first removal of the elongated process. Kulvin reviews the clinical significance of this condition: First, in the difficulty of diagnosis from sensation standpoint between this and the presence of foreign body; second, in the possible complications during tonsillectomy, incising of peritonsillar abscess or insertion of needle for suturing bleeding point, and third, in its occasional snug position between the carotids when ligation of the latter be necessary.

Fig. 5. Stump of styloid process after removal of fragment. Process on the opposite side also seen.

The normal styloid process is about 30 millimeters in length (one and one-fourth inches). Guthrie[7] quotes Gruber's results in the examination of 2,000 skulls in which only eleven styloid processes measured over one and one-half inches, and only one over three inches. The styolid process is a part of the hyoid bar of the second visceral arch of the embryo and consists of two parts, of which the first joins the petrous at or about the time of birth. From the cartilage of this second gill arch are formed the stapes, styloid process, stylohyoid ligament, lesser cornu and part of the body of the hyoid bone. The second part, according to Alexander,[8] represents all but the base as an ossification of the stylohyoid ligament and does not join the first part until puberty or later. An unduly long styloid process is the result of the ossification of this stylohyoid ligament. According to Kohler,[9] defective ossification of the styloid process, or rather of its two portions, is frequent, though complete ossification of the stylohyoid ligament sometimes occurs. Such an ossified stylohyoid ligament has often been mistaken by surgeons for a swallowed denture with opera-

tion for removal. Sir William Turner[10] reported a hyoid apparatus in man in which a separate epihyal bone was developed—the right styloid process 31 mm. in length, two or three times the normal thickness, instead of terminating in a point, expanded and formed an articulation with a bar of bone 56 mm. long, which passed down to articulate with the lesser cornu of the hyoid bone. The place of the right stylohyoid ligament was taken by this epihyal bone.

There does not seem to have been great difficulty in recognizing either an elongated or misplaced styloid process. Palpation upon localized area of tenderness should be sufficient, even without roentgen examination. In treatment the removal of the tip has been easily accomplished, but has often been left undisturbed, in the reported group. Rather curiously convalescence without removal has usually occurred. Bacher,[11] who reported sixteen cases of elongation complicating tonsillectomy found in the literature up to 1922, including two of his own, made this valuable diagnostic observation. "The characteristic movements of the styloid, in distinction from other possible bony substances, are a movement with the rotation of the head and movement with nodding of the head."

Operation upon the process may open up infective pathways in the retrotonsillar area. A classic example is that of Von Eiken,[12] in which infection of the petrous bone followed removal of a portion of the styloid process. A bony sequestrum was subsequently removed on continuance of symptoms. This was followed by middle ear complications, incision of tympanic membrane and finally involvement of the facial canal and petrous tip requiring major surgery.

REPORT OF SPECIAL CASE.

This patient, Miss A. W., 53 years of age, tall, rather thin, with a poor physical background, including suggestion of former pulmonary lesion, came to the office in 1916 because of a chronic discharge of the left ear. Her tonsils had been well removed by another physician some months previous. As it was impossible to pass a eustachian catheter upon the side of the offending ear, on account of septal deviation, a submucous resection was done. She

returned for further treatment in August, 1919, and in January, 1920, a simple mastoid operation was done, and patient discharged after a normal period. By midsummer there was a marked recurrence of mastoid symptoms with breaking down of the postauricular wound. This was accompanied by vertigo, discharge from the canal and behind the ear, with marked swelling and edema of tissues about the ear. A very extensive radical mastoid was performed in August, 1920, with unfavorable results. There was marked increase of labyrinthine symptoms and a temporary facial palsy. The bony structures about the ear were softened and carious, almost osteomyelitic in type, and a very extensive removal was required. Inadvertently a portion of the lower anterior wall was grasped and crumbled away, exposing the parotid area. This is quoted because of its possible etiologic bearing upon the subsequent fracture of the styloid process. An argument against this is the fact that the fracture was not at the base of the styloid —a very appreciable fragment will be noted in the slide—yet there may have been septic entrance into the styloid area, weakening the bony structure.

In the roentgenograms recently taken the softened condition of surrounding bone structure may be noted. This patient has been under occasional routine observation during the interim since the radical operation. Although she had a middle ear abscess on the right (opposite) side with resultant intermittent discharge and also required right ethmoid drainage some years after this radical mastoid, she had made a fairly complete recovery; vertigo and palsy disappeared, the ear was dry, and she carried on her duties in a responsible position in a department store.

In 1929, ten years after the last mastoidectomy, she complained of increasingly annoying throat symptoms in the left tonsillar fossa. This appeared to be more in the lingual tonsil or at the junction line of the base of the tongue. This was watched for two or three years, and small masses of lymphoid tissue were cauterized or removed. The pain, however, persisted and a slight protrusion in the left tonsillar fossa was noticed. X-ray films were taken by Dr. Pfahler, demonstrating the fracture of the styloid near the base. He has courteously furnished two other slides, one

showing a definite fracture of the left styloid process (case No. 1), and the other showing congenital failure in the ossification of the styloid ligament. On April 28, 1932, with the co-operative assistance of a general surgeon, Dr. Knox, the distal fragment was removed. This did not prove to be as simple as indicated in published cases, and consumed, partly because of ether complications, nearly two hours of excavation in the tonsil fossa. The segments of bone, which show how small this styloid projection was, are presented for inspection of the Society. This patient made a slow but uneventful recovery. The large aperture in the tonsil fossa is completely closed, there is no drainage or referred ear symptomatology. She still, however, complains of occasional periods of local soreness, rather neuralgic in type, but the throat appears normal.

CONCLUSIONS.

1. The group of published cases of fracture of the styloid process without obvious traumatic etiology is so small that it suggests a much larger unreported list.

2. The symptomatology in cases of fracture of the styloid process closely resembles that of simple protrusion of an elongated process.

3. Faulty ossification may readily account for the occurrence of both of these complicating disturbances.

4. It is of interest to note that removal of the offending tip has not always appeared necessary. The procedure in removal should not be attended with danger.

5. The complicated mechanism of the stylohyoid apparatus should readily permit the malposition of an elongated process. A minor deviation from the normal might explain long continued discomfort in deglutition customarily assigned to other causes.

1912 Spruce Street.

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LIX.

ACCIDENTAL PERFORATION OF THE ESOPHAGUS: GASTROSTOMY, RECOVERY—A PLEA FOR RETROGRADE DILATATION IN SMALL SIZED STRICTURES.*

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Small sized strictures, measuring 15 French or less and of some chronicity, are best treated with a greater degree of safety and permanency of cure by retrograde methods.

One might slightly modify the above statement by stating that should the stricture even be larger but considerable ulceration be present near the stricture because of food retention and decomposition, gastrostomy and retrograde methods should be used. The esophagus may be cleared of food in adults by drinking water at an interval after eating, but in children it is rarely done and ulceration just above the stricture is very common.

The aphorism of Hunter, so often quoted by Jackson, that "the patient with stricture of the esophagus rarely dies from it but dies from the bougie" is just as true today provided proper precautions are not followed.

The esophagoscope cannot guide one beyond the stricture, except a very short distance. The roentgenogram helps materially, but there is still a great element of danger in any method that is not guided by a string that may pass through the stricture and eventually be recovered in the stomach.

The introduction of piano wire guides is very dangerous, even when one feels that the additional safety of the fluoroscope is resorted to. A stricture so small that a piano wire guide can only be admitted, will permit a twisted silk thread to pass through

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*Presented at the meeting of the American Bronchoscopic Society at Washington, D. C., May 10, 1933.

From the Laryngological Service of the New York Post-Graduate Medical School and Hospital, Columbia University.

and without the ever present danger of perforation of the esophageal wall by the stiff wire.

The string method of Abbe with the retrograde bougies devised by Gabriel Tucker is used. Tucker's bougies are made of rubber and attached at either end to the string guide and may be left within the stricture for a period of time. They are extensible to a slight degree and when relaxed become slightly larger.

The method that has been used is to obtain the greatest dilatation with the least amount of pull and then leave the next size dilator within the stricture for from a half to one hour.

Rapid dilatation or increasing the sizes of the bougies too frequently should not be practiced.

The following detailed case report will serve to illustrate:

H. C., aged 20 years, was admitted to the New York Post-Graduate Hospital, July 8, 1927. Seven weeks previously he had swallowed acid from a storage battery, mistaking it for whisky. Was taken to the Jamaica Hospital, where his stomach was washed out several times and he was discharged within a week.

Within three weeks he began to have difficulty in swallowing. At present he has great difficulty in swallowing even water. Has lost thirty-eight pounds since the accident.

A roentgenogram showed a stricture at the lower third of the esophagus. Gastrostomy was done July 11, 1927. Within ten days bouginage on a string guide, using Plummer's dilator, was commenced. Esophagoscopy showed a stricture of 12 French at 37 cm. from the upper teeth.

Ten dilatations were done over a period of two and one-half months. He was able to swallow very well. The patient then was lost track of and did not report until July 11, 1932. During this interval he said that after five months the gastrostomy closed spontaneously and that for almost three years he experienced no difficulty in eating anything.

For the past two years he had been passing a rubber bougie into his esophagus before each meal. The day of admission he had been eating meat and swallowed an unusually large piece and it became lodged in his esophagus.

He was readmitted and following the removal of the piece of meat, the operator attempted to dilate the stricture that he saw through the esophagoscope and was of about a 20 French in size.

During this procedure the esophagus was perforated with an esophageal bougie, passed through the esophagoscope. The patient did not complain of any pain and a roentgenogram was taken that showed extravasation of the contrast mixture in the region of the constriction, suggesting a perforation.

I was communicated with and advised no further examinations or swallowing of any liquids but requested that an immediate gastrostomy be done. This was done and the mediastinal inflammation cleared up within

two weeks and the patient's temperature receded. There was a basilar pleurisy on the right side.

Within a month a retrograde dilatation with Tucker bougies was started, and by March 23, 1933, a 40 French bougie could be easily passed through the stricture and a 7 mm. esophagoscope was easily passed into the stomach. The gastrostomy now was closed.

Comment.—It is felt, that, having had the strictured esophagus for five years and having traumatized the viscus three times daily with a bougie for two years before the perforation occurred, the mediastinum was not vulnerable to diffuse infection.

The immediate gastrostomy, putting the esophagus at rest and the attempt by posture to keep as much saliva out of the esophagus would seem to have been some of the contributing factors that induced a recession in the mediastinitis. There were two reasons for keeping the saliva out of the wounded esophagus. The first, lessening the number of mouth and pharyngeal bacteria, and second, lessening the action of the saliva on the wounded tissues.

The diagnostic barium that extravasated into the mediastinum did not seem to further add to the infection.

It is necessary to keep in contact with patients who have or have had a stricture of the esophagus and have them report at regular intervals. I have found this to be a difficult matter—even with intelligent patients.

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LX.

EMPYEMA OF THE PETROUS APEX: FURTHER OBSERVATIONS AND CASE REPORTS.*

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Since the publication of our original series of papers[1] on this subject we have had eight additional cases. Of these, one has already been published by one of us.[2] All of these cases have thrown additional light on our topic and have served to corroborate our original observations.

Diagnostic Points.—It seems that we must further subdivide the cases of empyema of the petrous apex into two types, the acute and the chronic.

The acute cases present the symptomatology already outlined by us: namely, retro-orbital pain and profuse aural discharge. In addition, these cases run a clinical course ultimately terminating in meningitis unless surgical intervention is prompt. This type of case is easily recognized and may or may not be accompanied by an external rectus palsy. In any event, the presence of an external rectus palsy is not an essential to the diagnosis of empyema of the petrous apex.

At the time of our original publication we made the following statement: "We accept Wittmaack's work as the only logical explanation of hitherto unexplained observations in cases of chronic otorrhea. Nevertheless, his work does not account for the presence of a chronic suppurative otitis media associated with a normally pneumatized mastoid process wherein the squamous epithelium plays no rôle in either the cause or the cure of the lesion." At that time we had only one case to substantiate our belief that an empyema of the petrous apex could be a cause for a chronic otorrhea. Several of the cases herein reported definitely show that such is the case. In all of these, after well performed

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simple mastoidectomies upon well pneumatized mastoid processes, a chronic otorrhea resulted; and in spite of one or more revisions of the simple mastoid operation the chronic otorrhea remained uninfluenced and persisted. When operated on by us, all these cases were found to present a large fistulous opening into the petrous apex. The performance of the radical mastoid operation and the curettement through the fistula of the reachable pathology in the petrous apex resulted in a cessation of the discharge and a cure of the lesion. All of these chronic cases did not present retro-orbital pain; nor did they all present signs that might lead one to suspect the presence of an acute empyema of the petrous apex.

Diagnosis in these cases is made on the following clinical and laboratory findings: (1) Persistent otorrhea following simple mastoidectomy on well pneumatized mastoid processes wherein the causative factor was not an acute necrotic otitis media (scarlet fever, diabetes, etc.) and wherein the perforation in the membrana tympani was not marginal in type; (2) the failure to find any further pathology within the mastoid process on revision of the original operation; (3) the positive finding on roentgen examination of the destruction within the petrous apex.

Where such cases are associated with occasional attacks of retro-orbital pain, the diagnosis can be made from the clinical findings alone. However, where the eye pain is absent, the roentgen examination becomes the essential factor in diagnosis in any case of a chronic otorrhea not due to either an acute necrotic otitis, a transformative type of chronic otorrhea or a poorly performed technical procedure on the mastoid process.

Roentgen Findings.—We obtain the greatest amount of information from the employment of the base plate. Recently, however, this has been supplemented by taking a plate in the oblique position, known as the Stenver's position. The literature, within the past two years, has contained several reports of cases wherein the Stenver's position was of diagnostic import in cases of empyema of the petrous apex.

In this series of cases the Stenver's position was employed in many of them, in addition to the base plate, but it was found to be of value for diagnosis only in demonstrating a destruction of

the superior petrosal surface. The fine detail of cellular structure, which is evident on a well taken base plate, does not show up as clearly when the Stenver's position is used. We therefore recommend that the base plate be used for the interpretation of fine structural changes in the petrosal pyramid and that Stenver's position be employed only as a supplement to it.

As Taylor[3] points out, a clouding of the petrous apex will be evident in every case of purulent otitis media, just as a clouding of the mastoid cells will show up in every case of an acute purulent otitis media. The roentgen findings, therefore, should be considered diagnostic only where a destruction of cell wall substance is evident.

Surgical Therapy.—A great deal of misunderstanding has resulted following our original publication as regards the surgical therapy advocated for the cure of empyema of the petrous apex. The general impression seems to have been that we have advocated the Almour technic for all types of such cases. Because of this misconception we again reiterate our original standpoint.

Where there exists the symptom complex characteristic of an encapsulated empyema in the petrous apex, surgery should be employed for the relief of the deep-seated suppurative focus within the petrous apex.

Where, in addition to the above, the roentgenologic examination shows that the apex has retained its contour, the surgery employed should be one of the two following procedures:

A. If a pathway into the tip is demonstrable after a radical mastoidectomy has been performed, this fistulous tract should be cautiously widened, carefully curetted and probed to its depth. Where a flow of pus follows these procedures, the tract should be drained with a thin piece of rubber dam for a few days and then injected with lipiodol. By doing the latter we are afforded an excellent photograph of the tract into the petrous tip and, in the cases wherein we have used this procedure, it has had a curative effect in completely sterilizing the suppurative tract and focus in the pyramid.

B. If, after probing the tract, there is a failure to obtain a flow of pus, or in the event of being unable to discover the pathway of entrance, the technic described by us for drainage of the

petrous tip should be employed to evacuate the pus within the petrous apex.

Where, in addition to the clinical picture of the petrous pyramid suppuration there are other signs leading to a diagnosis of an epidural abscess in combination with it, or where the radiographic examination reveals a break in the contour of the petrous apex, this type of epidural abscess and the lesion in the pyramid should be drained by the subdural route, employing any one of the methods described by Striet, Goris, Broeckaert, Eagleton or Smith.

Where, after simple mastoidectomy on a pneumatic bone wherein the causative factor was not an acute necrotic otitis, and wherein there has been no secondary cholesteatome formation, a chronic otorrhea results, and where, in addition, the roentgenogram shows definite evidence of disease in the petrous pyramid, here the surgical technic described by us should be employed to eradicate the disease in the petrosa which we now know to be the cause of the chronic otorrhea in such cases.

It will be noted that we advocate the approach of the lesion in the petrosa through the wall of the tympanic orifice of the eustachian tube only in the event of being unable to find a fistulous tract opening into the petrous apex; in other words, where the pus within the apex is enclosed. In the cases reported by us herein, various types of procedures were employed in order to reach and to eradicate the lesion within the petrous apex. Where fistulæ were found, they were widened and curetted. Where no fistulæ were found, the petrous apex was entered through our avenue of election, at the mouth of the eustachian tube, directly beneath the tegmen tympani, after removal of the tensor tympani muscle.

Considerable discussion has arisen as regards (1) the logic of the Almour technic; (2) its safety; (3) its efficiency to consummate the eradication of the lesion.

We are now more firmly convinced that our procedure is the rational and natural approach to the petrous apex, first, because from the pathologic studies of Lange, it has been shown that the pus within the petrous apex usually finds egress and drains through the peritubal cells; secondly, our own observations at the operating table show that where spontaneous fistulous open-

ings are found, they are usually located at the mouth of the eustachian tube at exactly the point which we select for making our entrance into the petrous apex; and thirdly, from the fistulous tracts found, when we estimate their direction against the axis of the external auditory canal, they are found to make an angle of 22 to 25 degrees, the angle opening posteriorly—the angle used in the technic we advocate to guide our burr in entering the pyramid.

As regards the safety of the procedure, the cases which we have operated upon have shown (1) no purulent labyrinthitis; (2) no destruction of cochlear sound perception; (3) no carotid bleeding; (4) no meningitis.

As regards the efficacy of the surgical procedure, all our cases have recovered in the last series, amounting to seven, reported in detail below.

Anent the statement which we originally made and which we have reiterated above, that where a radiographic examination reveals a break in the contour of the petrous apex, the epidural abscess should be drained by the subdural route, we call attention to case No. 1, wherein such a break did occur and wherein subsequently the injected lipiodol spread intracranially between two layers of the dura forming the tentorium from the region of the anterior cranial fossa backward almost to the internal occipital protuberance, and wherein adequate drainage was obtained for the pus within the petrosa and subdural abscess and a complete recovery resulted without operation by the subdural route. If the subdural route had been selected in this case the lesion would not have been reached.

Since the publication of our original report several other methods of operation have been presented in the literature. It is proposed to discuss these fully at another time. Here it suffices to say that each one of these technics is adequate only in certain specified types of cases, just as our own technic is limited in scope to lesions reachable by it. However, the facility with which the lesion is reached, using the technic proposed by Almour, and the adequate results obtained by draining an encapsulated abscess within the petrous apex wherein no fistula has been found, com-

mends this procedure as the ideal method of approach in the acute type of case.

As in case No. 5, wherein a fistulous tract was found present in the epitympanic space, the performance of the radical mastoidectomy became unnecessary for the cure of the empyema in the petrous apex. In operating upon such cases we again caution the surgeon to make careful search for the fistula, with the hope that, in a few instances at least, the conversion of the simple mastoid wound into a radical mastoidectomy may be obviated, because the fistula will have been found and the surgical objective reached.

Where, after removing the outer attic wall and before the posterior canal wall and the middle ear have been demolished, a fistula is found present in the epitympanic space, probing and curetting this fistula may suffice to clear up the lesion. If such procedure does not result in a dry middle ear and a healed mastoid wound, it then becomes necessary to extend the limits of the surgical therapy, and to perform a radical mastoidectomy coupled with drainage of the petrous apex through the mouth of the eustachian tube, to establish cure.

Operative Findings.—From our knowledge of pathologic findings in acute middle ear and mastoid involvements, the middle ear does not usually present detritus and granulation tissue. There is often seen, especially in children, an edematous thickening of the lining mucosa.

Where we have been called upon to open the middle ear spaces in these cases of suppurative involvement of the petrous apex, we have invariably found the middle ear filled with granulation tissue and purulent detritus. This was found most marked in that type of petrous tip involvement which presented fistulæ.

From these findings at operation, we believe we are justified in holding that petrous apex suppuration is to be considered a complication of tympanic cavity suppuration, rather than a complicating lesion secondary to mastoid invasion.

Postoperative Course.—The severe retro-orbital pain complained of in the acute cases disappears within from twenty-four to seventy-two hours after drainage has been established. The acute cases of empyema of the petrous apex will, in the after-

treatment, show a dry ear within a very short time and epidermization of the radical cavity proceeds quickly. In the chronic cases, however, wherein the predominant symptom is the persistent otorrhea, the drying up of the radical cavity takes a much longer time. Granulations tend constantly to spring up in the region of the tube, and a dry ear will not be obtained unless these granulations are burned off daily, and adequate access afforded the pus within the petrous apex. The case is considered cured when there is no more discharge and epidermization is completed.

CASE REPORTS.

ACUTE TYPE.

Case No. 1.—J. B., 22 years of age, was referred to us by Dr. Nash of Rochester, N. Y., on August 29, 1932, with the following history:

On June 3, 1932, she had a streptococcus sore throat. Severe pain in the right ear set in on June 5th. A myringotomy was performed on that date and again four days later. On June 12th, a right simple mastoidectomy was performed by Dr. Henry Tollman, Jr., of Salem, Massachusetts. The findings at that time showed an extensive necrosis of the mastoid cells and the organism recovered was a streptococcus. Convalescence from then on was very slow; the wound did not heal well and granulations were very slow in forming. On August 3rd, Dr. Tollman again opened the wound and found two sequestra which he thought accounted for the persistence of the otorrhea.

Immediately following this second procedure the patient began to complain of severe pain behind the right eye and over the right side of the head. She was examined by Dr. Randall, a neurologist, who reported her negative from a neurologic standpoint. On August 23rd she complained of diplopia. Examination of the eye grounds at that time gave negative results. Her temperature was normal until August 25th, after which there was a daily rise to 102° F.

The diagnosis of empyema of the petrous apex was made by Dr. Nash and the patient was brought to New York and taken to Beth Israel Hospital.

Examination at the hospital revealed an open right mastoid wound which showed a moderate amount of pus coming from the antrum. The right middle ear was filled with a thick discharge which when wiped away immediately reappeared through a perforation in the antero-inferior portion of the drum. There was a distinct paresis of the right external rectus muscle. The patient was in agony from the severe pain she complained of behind the right eye. The fundi were negative. There was definite cervical rigidity. The temperature was 103.2. An X-ray of the temporal bone was taken in the usual manner and also one in Stenver's position and a base plate. These revealed a definite break in contour of the superior surface of the petrosal pyramid, indicating a rupture at that point. (See Fig. 1.)

Fig. 1. On right side definite break can be seen in continuity of superior petrosal surface. Entire contour of tip completely destroyed. Compare with left side.

The diagnosis being substantiated, this patient was operated upon on the day of admission. The skin edges of the old wound were retracted and the inner table of the mastoid exposed. The root of the zygoma was next removed. The wound was then converted into a radical cavity by lowering the posterior meatal wall and removing the outer attic and antral wall. The anterior buttress formed by the zygomatic root was then completely thinned down with a burr and the mouth of the Eustachian tube was brought into full view. A large area of necrosis was then seen in the tegmen tympani just above the mouth of the Eustachian tube. This necrotic area extended inward toward the petrosa and was followed for a depth of ¼" and removed. The tensor tympani muscle was next removed and the petrous apex was entered with a drill held at an angle of 22° to the external auditory meatus. The petrous apex was found to be one large cavity filled with sickly granulations. Two cc. of lipiodol were next injected into the petrous pyramid. The wound was left open and packed with rubber dam.

X-rays, both lateral and base plates, were then taken and showed the petrous apex to be filled with lipiodol. There was an extravasation of a small quantity of lipiodol along the lateral portion of the petrous bone. In addition, there was an extravasation of lipiodol along the inner border of the petrous bone which extended posteriorly above the tentorium toward the internal occipital protuberance. (See Figs. 2 and 3.)

Immediately after the operation the temperature rose from 103 to 104 and then fell to 102, varying between this point and 101.2 for the next few days. The opisthotonos gradually cleared up and the severe retro-orbital pain completely disappeared the second day after operation. The diplopia persisted for four days and then cleared up. The external rectus palsy was completely gone by the end of a week.

During the second post-operative week there was a sharp rise of temperature to 104, unaccompanied by any other symptoms. Inspection of the wound revealed a large amount of discharge coming from the opening into the petrous apex. Blood culture was sterile. Repeated small transfusions were then given. The temperature gradually returned to normal and at the end of five weeks the plastic operation was performed on the external auditory canal and the wound was closed.

The patient was discharged from the hospital on October 5, 1932, and the radical wound was treated at our office. At the present writing the wound is completely dry and the patient has fully recovered.

COMMENT.

In this case, one of the acute type, we suspected prior to operation, that a rupture through the superior surface of the petrous apex had occurred. Nevertheless, we were able to successfully drain both the apex and the intradural collection of pus through the method we employ to open into the petrous tip.

Case No. 2.—S. G., age 7 years, was taken ill on January 17, 1931, with pain in both ears. Bilateral paracentesis was performed on the following day. The temperature rose to 106 on January 23rd and the child was admitted to the Beth Israel Hospital for observation.

Fig. 2. Immediately after operation and injection of lipiodol into petrosa. Large intracranial collection of lipiodol seen.

Fig. 3. X-ray taken September 30, 1932, shows extent of extravasation of lipiodol.

Examination revealed definite signs of an acute coalescent mastoiditis in the left ear and a receding acute purulent otitis media in the right ear.

On January 26th, a left simple mastoidectomy was performed by Dr. J. C. Scal. The findings were those characteristic of an acute coalescent mastoiditis. The pneumatization was extensive. The child was discharged from the hospital with normal temperature on February 1, 1931. The post-operative course was uneventful and the child fully recovered, with a completely healed middle ear and mastoid wound.

In about the middle of November, 1931, the boy began to complain of severe pain in and behind the right ear. Temperature rose to 102 and the child was admitted to the Manhattan Eye, Ear and Throat Hospital, where a right mastoidectomy was performed.

Twenty days following the operation, the child was apparently well and then suddenly, the temperature rose to 102, and the right ear began again to discharge profusely. The mastoid wound did not heal. Periodic attacks of vomiting set in. A transfusion was performed and the child apparently recovered from this attack.

One and a half weeks before the second admission to the Beth Israel Hospital, the boy began to complain of right temporal headaches, vomiting, moderate amount of fever and pain in the right eye. He was admitted on January 20, 1932, with the diagnosis of acute empyema of the right petrous apex.

X-ray of the left mastoid showed signs of previous operation. The left petrous pyramid was pneumatized and normal. The right mastoid showed no pneumatization. There was a large area of cortical destruction in the mastoid proper, which extended into the squamosa and was due to operative interference. The sinus wall was situated well forward and was intact. The upper portion of the sigmoid sinus and knee showed considerable decalcification. There was a dehiscence in the dural plate near the knee of the sinus. There was a small emissary vein present, a short distance below the knee of the sinus. The X-ray showed a generalized diminution in aeration on the right side. There were a few small pneumatic cells present, posteriorly and external to the labyrinth. There were no cells present internal and anterior to the labyrinth. The petrous apex showed an intense atrophy with a loss in trabeculations. There was noted a slight irregularity in the anterior border of the petrous bone near the apex. There was another localized irregularity in the contour of the petrous bone at the anterior end of the inferoposterior border near the sinus. The right petrous apex showed changes associated with petrous pyramid suppuration. (See Fig. 4.)

On January 21, 1932, an incision was made through the old mastoid scar on the right side and the wound edges were separated. The mastoid cavity was found filled with infected granulation tissue and when this was removed the exposure of the sinus noted on the X-ray plate was identified. The cavity was next converted into a radical one by breaking through the posterior meatal wall and lowering it. A thorough toilette of the middle ear was performed and the infected, boggy granulations were removed. The attic and aditus were filled with infected granulation tissue. When the latter was removed, an area of necrosis was seen above the horizontal canal and behind the superior canal. When

Fig. 4. Right petrous apex shows changes due to an intense atrophy and loss of trabeculations as compared with opposite side.

a probe was inserted into this fistula it took a general direction first inward and downward, and then inward and forward for a depth of two inches. The end of the probe touched bone. This fistulous opening was enlarged and the probe gently manipulated inward and outward. This procedure was followed by a gush of approximately two drams of thick, creamy pus. Rubber dam drain was inserted into the opening and the wound packed with rubber dam.

On January 25, 1932, an X-ray of the petrosa was taken with a probe inserted into the fistulous opening. This showed the tip of the probe to be near the region of the inferior petrosal sinus, extending to the lower anterior portion of the petrous apex. The direction of the probe was into the upper portion of the petrous bone, above the auditory canal, extending forward, inward and downward, terminating in the anterior portion of the petrous apex in the region of the inferior petrosal sinus. (See Fig. 5.) Injection of lipiodol through the fistulous opening showed a small collection of lipiodol in the upper and posterior portion of the petrous apex and a tract of lipiodol above the labyrinth, extending in the direction of the apex.

On February 1, 1932, a plastic flap was made and the wound was sutured. Following this the discharge continued to be profuse and the wound broke down. A low-grade temperature set in, running up to 101. The blood count, which on admission showed 4,420,000 red blood cells with 65% hemoglobin and 15,800 white blood cells with 83% polys and 3% staff cells, had come down to normal only to rise on February 1st

Fig. 5. Probe inserted through operative area with point of probe at the apex of petrosa.

to 18,000 white blood cells with 84% polys and 3% staff cells. Careful inspection revealed the fistulous opening to be covered with granulations, thus interfering with drainage.

On February 23, 1932, the wound was again explored. The granulations were cleaned from the fistulous tract but no pus was found coming from it. It was then deemed advisable to make a counteropening into the petrous tip, using the Almour method of approach. After the zygomatic buttress was removed, the tympanic orifice of the Eustachian tube was identified and the petrosa entered with a burr held at an angle of 20° to the external auditory canal. Both the fistulous tract and the tubotympanic opening into the pyramid were drained with two strips of rubber dam.

A transfusion of 150 cc. of blood was given on February 26th.

The first dressing was performed on February 27th and a profuse purulent discharge was noted coming from both tracts leading into the petrous apex. The temperature, which had assumed a mild septic course on February 17th and from then on varied between normal and 102 and 103, came down to normal on March 1st and has remained so. The blood count returned to normal by March 4, 1932, and the discharge from the petrous apex markedly diminished. From the time of admission until the patient was discharged, on March 8th, no more eye pain was present. The boy was dressed in the office until all discharge from the middle ear had ceased.

On October 28, 1932, a plastic operation was performed on the radical mastoid wound. The thin scar tissue of the posterior lip of the wound

was cut away from the remainder of the lip with scissors, leaving only a pedunculated attachment to the upper angle of the wound. The under surface of this area was curetted and made to bleed and was then laid into the radial wound so that the distal extremity of the flap covered the inner wall of the middle ear and from then outward to its attachment, covering the tegmen tympani, tegmen antri and exposed dura. Kerr's impression-compound was then inserted into the wound so as to completely approximate the flap to the walls of the middle ear.

On November 16, 1932, the posterior wound was closed. The anterior wound flap was carefully dissected away from the underlying conchial cartilage for a considerable distance, thus affording a sufficient amount of mobilization so that approximation of this flap with the posterior wound edge was possible without tension. The flap of skin previously thrown into the middle ear was now severed at its attachment to the skin, and the posterior wound edge was bevelled off so that a new broad, raw surface was created. In this manner also a new skin edge was created. The anterior lip of the wound was now sutured to the skin margin by interrupted silk sutures.

At the present time the child has made a complete recovery.

COMMENT.

1. This is a case of the acute type of empyema of the petrous apex. The severe eye pain, fever, profuse otorrhea and transient labyrinthine signs were all present.

2. In this instance, drainage of the fistula did not in itself suffice for the cure of the lesion. All signs disappeared and the discharge ceased only after a counter-opening into the tip was established at the mouth of the eustachian tube.

Case No. 3.—L. J. D., age five years, had an attack of influenza in February, 1932. This subsided in two weeks, to be followed by a complaint of pain in the right ear. A right myringotomy was performed on March 10, 1932. The otorrhea persisted for two weeks. On March 24, 1932, a right mastoidectomy was performed. Following this, the child complained of intermittent pain in the right cheek which, except for a period of three weeks, was constantly present up to the time of admission to the hospital on June 15, 1932. This pain would last for an hour or two at a time and then be absent for the remainder of the day. Double vision was also complained of and the eyes would appear crossed at times. The drainage of pus from the mastoid wound was constant.

On admission to the Beth Israel Hospital the essential factors on examination were a scant discharge from the right mastoid wound, a profuse discharge from the right external canal, a slow nystagmus to the left on looking to the left, no ocular muscle palsy (despite the previous two attacks of diplopia), ocular fundi normal, temperature 100° F., no neurological abnormalities. The urine was normal. The blood count was 4,170,000 red blood cells with 57% hemoglobin and 17,500 white blood cells with 78% polys and 2% staff cells.

Roentgenographic examination of the nasal accessory sinuses and temporal bones on June 16, 1932, showed the mucous membrane of the sinuses to be congested. The right mastoid process was post-operative and showed no cellular remains; no sequestra were present. The left mastoid process was normal. The left petrous pyramid was normal. The apical portion of the right petrous pyramid showed absence of trabeculae, decalcification and atrophy. The apical contour was intact. The appearance of the right pyramid was that of a suppurative lesion of its apex. (See Fig. 6.)

Because of (1) the persistence of an otorrhea following a simple mastoidectomy in a pneumatized process wherein the disease was not an acute necrotic otitis media; (2) and wherein the mastoidectomy was well performed; (3) and wherein evidence of trigeminal irritation was present;

Fig. 6. Loss of trabeculation and intense atrophy of right petrous bone.

(4) and wherein transient attacks of abducens palsy were present; (5) and wherein transient symptoms of labyrinthine irritation were evident, a diagnosis of empyema of the petrous apex was made. This was supported by the roentgen findings.

On June 17, 1932, the old mastoid wound was reopened and the granulations removed from the mastoid process. A necrotic sinus plate was found and also an exposure of the temporal dura. The wound was converted into a radical cavity in the usual manner. The middle ear was filled with granulations. These and the ossicles were removed. The mouth of the Eustachian tube was brought into full view by removing the zygomatic root in toto and thinning out the anterior canal wall. A fistulous opening was then noted to be situated on the inner wall of the middle ear at the Eustachian orifice. The tensor tympani muscle was

next removed. A probe was inserted into the fistula and this took a direction inward and forward toward the petrous apex. This opening was enlarged with a burr and the petrous apex could then be easily entered. This was found to be a large cavity filled with soft, mushy granulations. Careful probing of this cavity could elicit no break in the inner table. All further procedure was then stopped. The cavity in the petrous apex was drained with rubber dam and the wound was packed with iodoform gauze.

The temperature postoperatively varied between 99 and 100, with but two rises to 101 (June 22nd and July 6th). Daily dressings were performed because of a profuse foul discharge from the wound. The white cell and differential count gradually returned to normal. On July 6th, the posterior wound was sutured and a Panse plastic performed on the posterior membranous canal wall. The child was discharged from the hospital on July 12, 1932.

COMMENT.

The opening of the fistula into the petrous apex was situated at the mouth of the eustachian tube at exactly the point which we select in making an entrance into the apex.

The transient nature of the diplopia is to be noted.

Case No. 4.—M. G., 33 years of age, was taken ill with influenza in December, 1932. In the second week of his illness his right ear began to

Fig. 7. Right petrous apex shows area of absorption in anterior and superior portion.

discharge. On January 21, 1933, a right mastoidectomy was performed by Dr. Melvin J. Stearns of Ogdensburg, New York. Following this operation he developed severe frontal pain and pain in the right eye. The discharge following operation continued and he failed to show any improvement. The post-auricular discharge was scant. The diagnosis of empyema of the petrous apex was made by Dr. Stearns and the patient was referred to us.

Otoscopic examination revealed a profuse purulent otorrhea on the right side and a slight amount of discharge from the lower angle of the mastoid wound. His temperature, on admission to Beth Israel Hospital on February 25, 1933, was 99.6. Blood count on admission was normal. X-ray examination of the temporal bones showed a post-operative right mastoidectomy. There was a definite area of absorption in the anterior and superior portion of the apex of the petrous bone on the right side, indicating a destructive change in the tip of the right petrous pyramid. (See Fig. 7.)

On February 25th, a right radical mastoidectomy was performed. The simple mastoidectomy was converted into a radical cavity. The structures in the middle ear were removed and a large number of granulations also curetted therefrom. In pulling out the tensor tympani muscle, a large fistulous opening was found at the mouth of the Eustachian tube which led directly into the petrous tip. A probe was inserted into this opening and took a direction forward and inward, making an angle of 23° with the axis of the external auditory canal. (See Fig. 8.)

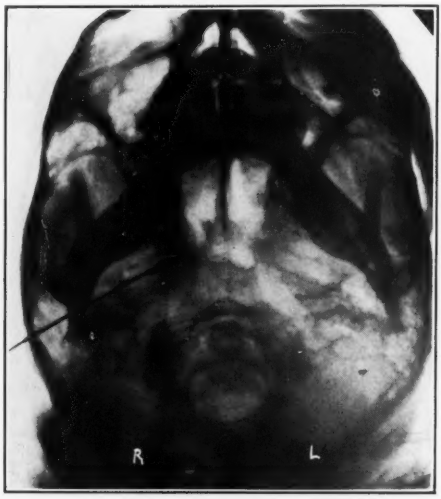

Fig. 8. Probe inserted through operative area with point of probe reaching apex of petrosa.

The post-operative course was uneventful. The retro-orbital pain disappeared within two days. The temperature, outside of the post-operative reaction to 102, remained normal. The patient was discharged from the hospital on March 9, 1933, and is still under observation and treatment and is progressing satisfactorily.

CHRONIC TYPE.

Case No. 5—A. C., 8 years of age, was admitted to the Beth Israel Hospital on June 28, 1932. Six years prior to admission the patient had had a bilateral mastoidectomy performed, following which he fully recovered. Three and one-half years later, the child developed a "pus bag" beneath one ear (which one not known); this was incised and healed within four weeks. The child was perfectly well until five weeks before admission, when he began to complain of earache and had an elevation in temperature. At the end of the second day of illness, one ear ruptured spontaneously and the other was paracenticized. One week following this, the left mastoid wound swelled and was incised; and a week afterward the right mastoid wound was incised. For the week prior to admission the temperature was elevated and there was a profuse discharge from both ears and both postauricular openings. The child complained of generalized headache. There had been no cervical rigidity, no pain in the face or in the eyes. There had been no chills.

Examination revealed a scant purulent discharge from both post-auricular wounds. Both external auditory canals were narrowed and contained a slight amount of pus. The narrowing of the canals was the result of the previous surgery to the mastoid processes. There was an early atrophic rhinitis. The blood count showed 4,100,000 red blood cells with 68% hemoglobin and 11,400 white blood cells with 66% polys.

Roentgenograms of the mastoid regions showed but a few small cells in the region of the mastoid tip on both sides; no sequestra were visible.

The child was kept under observation until July 2nd. During the stay in the hospital, the discharge from both wounds and middle ears greatly diminished; and the child was referred to an otologist for ambulatory treatment. The right side healed completely within a few weeks but the left wound, despite all treatment, continued to discharge. The child was then readmitted to the hospital on September 19, 1932.

X-ray examination of the petrosa on September 20th showed the right petrous apex to be normally aerated. The left petrous bone showed a slight diminution in aeration but no destructive changes. Dr. H. K. Taylor, in reporting the films, stated, "The diminished aeration of the left petrous apex may be due to an inflammatory lesion at this site which is the cause of the protracted otorrhea."

On September 21st, a revision of the left mastoidectomy was performed. Some necrotic cells were found in the squamosa. Lipiodol was injected into the antrum and some was left in the mastoid cavity. The wound was closed with three catgut sutures and drained with rubber dam.

There was a post-operative rise in temperature to 102.2 and a rapid return to normal the following day. X-ray of the petrosa on September 23rd showed a small collection of lipiodol in the petrous apex on the left side. (See Fig. 9.)

Dressings were performed daily. At each dressing, more and more pus appeared both in the external canal and from the mastoid wound. On September 30th, the wound broke open. By October 5th, the entire wound had broken down. The blood count during this period was normal.

In view of the roentgen findings and the continued suppuration, it was decided to explore the petrous apex. On October 7, 1932, the posterior wound was reopened and the skin edges retracted. The antrum was located. The outer attic wall was next removed so as to widely expose the aditus and antrum and the epitympanic space. Granulations were present on the inner antral wall and when these were removed a large

Fig. 9. Lipiodol injected into antrum at time of simple mastoidectomy found to have reached petrous apex.

fistulous tract was found leading inward and forward into the apex of the petrosa for a depth of 1½". This opening was situated above the horizontal and behind the superior semicircular canal. The orifice of this fistula was widened and the granulations removed by curetting. This area was then drained with a piece of rubber dam. The wound was left open. (See Figs. 10 and 11.)

For the next week, the temperature varied between 99 and 101, with but one rise to 102 on October 11th. On October 26th, a plastic repair of the mastoid wound was undertaken. This, however, was unsuccessful and the wound again broke down, due to a staphylococcus skin infection. The wound was then permitted to heal by granulations.

Fig. 10. Stenver's position showing lipiodol occupying petrous apex.

Fig. 11. X-ray taken on October 20, 1932.

During the remainder of the stay in the hospital, there were at times some slight temperature elevations to 101. At one time, November 28th, a mild attack of pleurisy set in. This disappeared within two days. The child was discharged from the hospital on December 12th, with but slight discharge from the wound. Dressings were continued in the outpatient department and at the present time the wound has completely healed and the middle ear is dry.

COMMENT.

In this case no radical mastoid surgery was done. The fistula into the petrous apex was situated in the aditus and consequently was identified by wide exposure of the antrum and aditus. Drainage of this tract resulted in complete cure without resort to any further surgery.

Case No. 6.—J. K., 5 years old, was admitted to the Beth Israel Hospital on May 14, 1932. Four weeks prior to admission the child was a patient at Gouverneur Hospital, where he was treated for a sore throat and measles. One week later, had onset of pain in the right ear and spontaneous rupture of drum, with continuous otorrhea since then. Examination revealed a circumscribed bulging of the posterosuperior portion of the right drum with a nipple perforation through which a pulsating discharge appeared. There was some tenderness over the mastoid process but no periostitis. The temperature varied between 99 and 100. The blood count showed 3,950,000 red blood cells with 56% hemoglobin and 13,000 white blood cells with 89% polys and 4% staff cells. The child seemed to improve and was then discharged to the outpatient department for treatment on May 18, 1932.

The child was readmitted to the hospital on May 25th, because the discharge became more profuse. Examination at this time revealed marked tenderness over the antrum and mastoid tip. Some periostitis was present. Roentgenograms of the mastoid processes and petrosa revealed a considerable decalcification and destruction of the bony trabeculae on the right side and a slight diminution in aeration of the right petrous apex. A simple mastoidectomy was performed on May 27th. Upon removal of the cortex, pus escaped under pressure. The mastoid process was entirely broken down and filled with pus and fresh granulations. The inner table was exposed throughout. The antrum was widely opened and the zygomatic cells exenterated. A portion of the mastoid tip was removed. The sinus was exposed and found normal. The wound was partly closed and drained with rubber dam. The culture of the pus showed streptococcus viridans. There was a postoperative rise in temperature to 101.8, followed by a return to normal the following day. The convalescence was uneventful beyond a slight stitch infection. The child was discharged to the outpatient department on June 4th, 1932.

One week after discharge from the hospital, the child began to have constant pain over the inner and upper part of the right orbit. The discharge from the middle ear became profuse. It was then noticed that the child had a strabismus and was unable to move the right eye laterally. The temperature was never higher than 100.2. The boy was readmitted

to the hospital on June 15th, with a profuse greenish discharge from the wound and from the middle ear. The drum was thickened and beefy in appearance. The fundi were negative. There was a total right abducens palsy. The blood count on admission showed 19,000 white blood cells with 72% polys and 4% staff cells. A roentgenogram on the 16th of June as compared with that taken of the petrosa on the 26th of May, showed a partial destruction and a moderate degree of atrophy; the findings were those of petrous suppuration. (See Figs. 12 and 13.) Successive X-ray examinations were made on June 20th, 22nd, 24th, 27th, 28th, 30th, July

Fig. 12. June 30, 1932. Right petrosa shows partial destruction and atrophy.

5th and 6th. All these showed no progression of the destructive process within the petrous apex. In the meantime the blood picture returned to normal and the temperature never rose above 100. The otorrhea rapidly diminished in amount and the abducens palsy began to improve on June 22nd and was entirely gone on July 1st. The improvement in both the paralysis of the external rectus and in the condition of the middle ear and mastoid wound was so marked that the attendant in charge thought it advisable to refer the patient to the outpatient department for dressings. The child was discharged from the hospital on July 9, 1932.

Despite frequent dressings the otorrhea persisted. The child was well otherwise. Because of the findings on roentgen examination and the persistence of the profuse mucoid stringy discharge from both the middle ear and mastoid wound, it was decided to explore the petrous apex in order to enlarge the fistulous opening into the apex from which it was felt that the chronic otorrhea emanated.

On August 10, 1932, the posterior wound was reopened and the soft parts retracted. The mastoid cavity was filled with sickly granulations which were removed. It was then seen that a tract of cells was present posterior to the vertical semicircular canal and that these extended inward toward the pyramid. These cells were necrotic and were accordingly removed. The cavity was next converted into a radical one and the contents of the middle ear eviscerated and the granulations removed therefrom. The tensor tympani muscle was next removed and the eustachian tube curetted. Upon clearing the area of election for entrance into the

Fig. 13. Stenver's position. August 6, 1932, complete destruction of right apex. Loss in continuity of superior petrosal surface.

petrous tip, a large fistulous opening was found present at this point. A probe inserted into this area passed easily inward and forward and, when calibrated with the external auditory canal wall, it made an angle of 22° to the axis of this structure. Gentle manipulation with the probe so as to break up the granulations within this cavity was immediately followed by a flow of about 2 cc. of thick yellow pus under pressure. Lipiodol was injected into this fistulous tract to the petrous apex; and at the first injection a flow of pus appeared. The culture of this pus showed streptococcus hemolyticus.

X-ray of the petrous bone showed the lipiodol to have reached the area in the right apex previously reported as having shown destruction. (See Fig. 14.)

The postoperative course was uneventful. The temperature never rose above 101 and this on only a few occasions. The blood count remained normal. On August 29, 1932, a Panse flap was cut in the posterior mem-

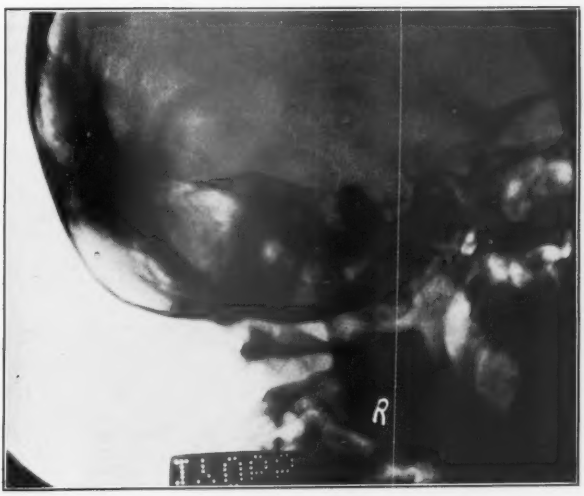

Fig. 14. August 10, 1932. Following operation and injection with lipiodol. Lipiodol seen to occupy entire petrous apex.

branous canal wall and the posterior wound was sutured. The child was discharged from the hospital on September 6, 1932. The radical cavity is now completely epidermatized and dry.

COMMENT.

A fistula was here found at the mouth of the eustachian tube at the exact point where we make our entrance into the petrous tip.

Case No. 7.—M. K., 8 years of age, had a bilateral mastoidectomy performed in 1929 and made an uneventful recovery. On March 7, 1930, she developed grippe and within four days had pain in the right ear. On the sixth day of her illness a right otorrhea appeared, with the temperature ranging between 100 and 104. The child was admitted to the Beth Israel Hospital on March 14, 1930, and was operated upon the same day. A revision of the right mastoidectomy was performed and free purulent fluid was found in the mastoid cavity. A few residual cells were found. The inner table was exposed throughout. The child made an uneventful recovery and was discharged on March 22, 1930.

In December, 1932, she developed a cold and a rise in temperature to 101.5. Bilateral otorrhea appeared within a short time and the temperature rose to 104. The child was admitted to the Beth Israel Hospital on December 31, 1932, temperature ranging between 101.5 and 102. She was kept under observation and the temperature dropped to normal on Janu-

ary 6, 1933. The blood count, which showed 12,700 leucocytes with 73% polys on admission, dropped to normal on January 6th. On January 8, 1933, there was noted a marked swelling over the left postauricular wound and pressure on this caused a profuse discharge from the external auditory canal.

On January 9, 1933, the patient was operated upon and the left mastoid operation revised. The entire mastoid process was found to be a hollowed-out cavity containing pus. The lateral sinus protruded prominently and was exposed for a small area. The wall was found to be slightly discolored and whitish. Further removal of the sinus plate revealed a healthy wall. The antral opening was exceedingly large. The wound was partially closed and drained with rubber dam.

Immediately after the operation the temperature rose sharply to 104.8 and during the following ten days gradually returned to between 99 and 100,

Fig. 15. January 23, 1933. Marked degree of osteoporosis and obliteration of left petrous apex.

with daily excursions to 102. The child had no complaints and was feeling well, but the discharge from the middle ear continued to be profuse.

Because of the continued profuse otorrhea, unaccounted for by any lesion within the mastoid process, and because of the acuteness of the lesion, we suspected that the source of the suppuration might be in the petrous apex. On January 23, 1933, a base plate of the temporal bones was taken and showed a marked degree of osteoporosis of the apex of the left petrous pyramid, with obliteration of the apex. The contoural outline was still retained. The changes involved the distal half of the petrous bone. (See Fig. 15.)

On January 27, 1933, the old wound was reopened and the cavity converted into a radical one. All granulations were carefully wiped away from the mastoid and antrum. The middle ear was found to be full of bleeding granulations and small polyps. The tensor tympani was removed and the mouth of the eustachian tube identified. Careful probing found a fistula to be present just posterior to the mouth of the eustachian tube and directly under the tegmen tympani. A probe, inserted into this fistula, entered directly into the petrous apex for a distance of two inches. The direction of this probe made an angle of 22° with the axis of the external auditory canal. On attempting to widen this fistulous opening, it was found that the burr ordinarily used to enter the petrous tip was by far too small and this burr sunk directly into the tip. Lipiodol was

Fig. 16. Immediately after operation and injection with lipiodol. Lipiodol seen occupying entire petrous apex.

next injected through the fistulous opening and the excess lipiodol which leaked out around the cannula returned with fluid pus. The wound was left open.

X-ray examination immediately after operation showed the injected lipiodol clearly visualized within the petrous tip and in the sinus extending outward to the operative wound. (See Fig. 16.)

The postoperative course was uneventful, the temperature returning to normal. On February 11, 1933, a plastic operation was performed on the external auditory canal and the wound was sutured. The patient was discharged from the hospital on February 14, 1933, and is still under treatment for the postoperative radical wound. The discharge from the middle ear has completely ceased; and the case is progressing satisfactorily.

SUMMATION.

From the foregoing we conclude that the involvement of the petrous apex in pneumatized bones takes two forms. It is either an acute lesion, threatening meningeal involvement, or it is a chronic type of invasion, which does not threaten meningeal infection.

The chronic type gives rise to a chronic otorrhea in a pneumatized temporal bone, the seat of an acute onset, not included in the acute necrotic type of Wittmaack.

The involvement of the petrous pyramid is a complication of tympanic suppuration and not a complication of mastoid involvement.

From all pathologic evdience at hand, and from the observed location of spontaneous fistulæ, and from the results obtained by using it, in both the acute and the chronic type of petrosal pyramid suppuration, the surgical technic we advocate (the Almour procedure) is adequate for the purpose of establishing drainage and curing cases of suppuration of the petrous pyramid which are located in pneumatized bones.

The operative technic does not damage the cochlea or injure the facial nerve or the carotid artery.

51 WEST 73RD ST.

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LXI.

TUBERCULOSIS OF THE ESOPHAGUS WITH REPORT OF A CASE FOLLOWING LYE INGESTION.*

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Tuberculosis of the esophagus is extremely rare. The esophagus is involved much less frequently than any other organ, in this disease. The collected statistics of Lockard[6] disclosed that the esophagus was found to be involved twenty-five times in 16,489 autopsies, while in a group of 14,980 patients with tuberculosis the larynx was found to be affected in 2,003.

Denonvilliers[2] reported the first observation at autopsy of a case of tuberculosis of the esophagus in November, 1837. Six years later Louis reported six cases which he encountered during 120 autopsies. In 1877, Zenker questioned whether esophageal tuberculosis had been proven to exist. Until recent years this disease was observed only at autopsy, and in 1909 Gottstein made the observation that tuberculosis had not up to that time been diagnosed through the esophagoscope except after previous biopsy.

Lockard's review, twenty years ago, is one of the finest articles written on the subject. He gives the following as channels of infection:

1. Direct inoculation of predisposed tissues.
2. Tuberculosis occurring with carcinoma.
3. Direct inoculation without previous injury.
4. Extension from pharynx and larynx.
5. Hematogenous infection.
6. Caries of the vertebræ.
7. Caseous mediastinal glands.
8. Unclassified—channel unknown.

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*Presented at the meeting of the American Bronchoscopic Society at Washington, D. C., May 10, 1933.

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Although bacillus laden sputum is being constantly swallowed by those with active pulmonary disease the relatively small number of cases indicates that the esophageal mucous membrane possesses an unusual resistance to infection by contact. Although it is difficult and at times impossible to prove, it is very likely that these esophageal ulcerations occur because of a break in the continuity of the mucosal surface. This would not apply to esophageal lesions caused by the penetration of extra-esophageal disease.

Most writers give the impression that tuberculosis of the esophagus occurs only in patients who have advanced disease elsewhere. In recent years, however, there have been two case reports which do not bear this out. Torek[9] reported the case of a male of 69 who complained of difficulty in swallowing and localized the point of the obstruction to the passage of food in the lower end of his esophagus. He was thought to have an early cancer. Esophagoscopy and biopsy were carried out by Oberrender. The report from the laboratory indicated that the lesion was tuberculosis. There was an old fibrotic lesion in the right upper lobe of the lung.

The patient of Paviot[7] and his associates was a male of 65, who complained of dysphagia for two months. He was not esophagoscoped. At autopsy a perforation into the trachea was noted. There was no evidence of a pulmonary lesion.

Tuberculous lesions of the esophagus have been known to perforate into surrounding structures. In Flexner's[4] patient, the lesion ulcerated into the left pleural cavity, while in Schmid's[8] case the perforation involved the posterior mediastinum and right pleural cavity. Ferrabouc[3] reported a case in which the ulceration extended into the aorta to cause death by hemorrhage.

Vinson and Dobson[11] reported a case of upper esophageal stenosis caused by a tuberculous lesion. They quote Guisez as having observed six cases with the esophagoscope. Guisez[5] distinguishes three forms of tuberculous lesions:

1. The secondary type—from lesions outside of the esophagus.
2. The ulcerous type—which is seldom seen because it occurs late in the disease.
3. Tumor form—rare, only diagnosed microscopically.

Tuberculosis of the esophagus occurs most frequently in adults but has been reported in the young. Vimtrup's[10] patient was 2 years old. The child refused to swallow for eight days and finally died of inanition. Tuberculous ulcers were found in the pyriform fossæ and two elliptical ulcers were found at the level of the tracheal bifurcation. There were numerous small lesions in the lower half of the esophagus. Microscopic study revealed tuberculous granulations and bacilli. This was no doubt a case of hematogenous origin. It is likely that many more cases of tuberculosis of the esophagus would be found in infants who succumb to hematogenous infection by tubercle bacillus. Tuberculous lesions may occur anywhere in the esophagus.

An interesting case in which tuberculosis was associated with cancer and syphilis was reported by Dean and Gregg.

Symptomatology: This condition may be free from symptoms and its presence may not be known until it is discovered at autopsy. This is particularly so in the types which are caused by penetration from without, from caseous glands or vertebral lesions. The lesions originating within the esophagus will cause varying degrees of dysphagia with or without pain.

Diagnosis: The X-ray examination may reveal an ulceration of type and extent which could not be interpreted as due to any other condition than tuberculosis. The fine moth-eaten ulceration in the case which is being reported is significant. Esophagoscopy is essential and should not be omitted. It would seem that the ulceration has a characteristic appearance such as is seen in laryngeal lesions, and for this reason esophagoscopic diagnosis should be made, in many of the cases.

CASE REPORT.

P. W., male, age 41, was referred by Dr. Ornstein for esophagoscopy. He had swallowed lye one and one-half years ago and had been treated until six months previously for the stricture which had resulted. He felt better as far as the esophageal stenosis was concerned, but his diet was restricted to soft and liquid foods. He was referred because he had recently developed a dull pain which he localized at the level of the lower end of his sternum.

Esophagoscopy revealed a stenosis of the upper esophagus twenty-two centimeters from the upper tooth mark. (Fig. 1.) On the posterior lip of

Fig. 1. Roentgenogram of the esophagus showing region of narrowing in upper esophagus where a tuberculoma was seen during esophagoscopy. Note moth eaten appearance all the way down.

this fibrous area there was a pale granulomatous mass which measured about six by three millimeters. This was diagnosed as a tuberculoma because of its similarity to lesions which the writer had seen in tuberculous larynges. The tumor was removed with punch forceps and submitted for histologic study. The pathologist reported tubercle formation in the submucosa. (Fig. 2.) Esophagoscopy was performed subsequently. Tissue removed at esophagoscopy was reported as chronic granulation tissue. At this time examination of the esophagus beyond the stenosis previously mentioned appeared to be superficially ulcerated as far down as the cardia, which could not be passed with a seven-millimeter tube. X-ray of the

Fig. 2. Photomicrograph showing a giant cell in the midst of an abundance of epithelioid cells.

lungs revealed a generalized fibrotic type of involvement with numerous small dilatations of the bronchial elements. The sputum was positive for tubercle bacilli. The intestinal tract was involved shortly before the patient died. Unfortunately, consent for autopsy was not obtainable.

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LXII.

PHYSIOLOGY OF HEARING—AURAL ACUITY—BONE CONDUCTION: A CRITICAL REVIEW.

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PHYSIOLOGY OF HEARING.

Up to a few years ago our knowledge of the physiology of hearing was based upon purely theoretical grounds. Since the work of Wever and Bray, Hughson and Crowe, Adrian and de No certain elements related to the manner by which we hear have been placed on a more factual basis. We know that the concha is useful in collecting the sound. When the external ear of the experimental animal is removed the intensity of the sound is diminished in the reproducer when the ear of the animal is rigged up as a microphone. The same thing happens when the external auditory canal is narrowed. The drum is not essential for the transmission of sound waves. Its complete circumcision does not reduce the intensity or clarity of the tones as long as the ossicles remain intact. The ossicular chain and the stapedius and tensor tympani muscles maintain and regulate intralabyrinthine pressure and act as safety devices for the prevention of injury to the membranous labyrinth by loud noises. When the continuity of the ossicular chain is broken all sounds stop except those from the higher forks and the higher notes of the Galton whistle. When the tension of the tensor tympani muscle is increased, all low tones are reduced in intensity. When the tension is removed, all sounds are heard with the same intensity as before the tension was applied. About nine days after the tendon of the tensor tympani muscle has been divided there is a loss in the ability to hear the high tones for 1024 D. V. upward, inclusive. One hundred twenty-eight D. V. is not heard, but 256 D. V. is quite distinct. After division of the tensor tympani muscle, if a pledget of cotton is applied to the round window, all tones are greatly increased in volume and clarity. This indicates that the

round window is a part of the mechanism by which proper intralabyrinthine pressure is maintained and adjusted. Puncture or rupture of the round window membrane results in a loss of transmission of all sounds.

From the research of Guild and his co-workers,[1] we know that the number of cells in the central region of the ganglion of Corti is greater than the number found at the apex and the base. It is therefore likely that the central portion of the scale is richer in the ability to transmit a greater variation in tone intensity and quality than that which pertains in the apical and basal regions. Lorente de No[2] has shown, as a result of his researches on the nerve tracts and connections of the auditory centers in the medulla, that a massing of a greater number of ganglion cells in the central portion of the spiral ganglion is represented in the ganglion ventrale of the medulla.

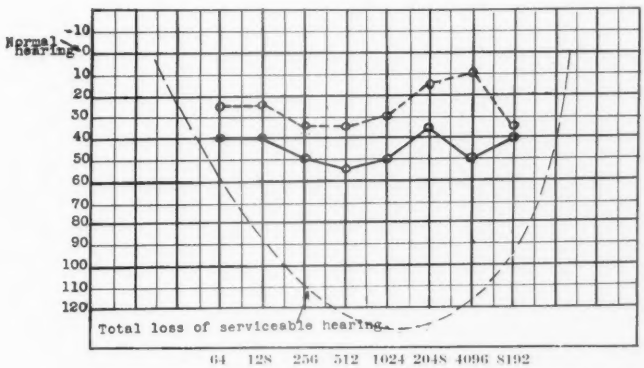
In addition to this, Guild made histologic examinations of the temporal bones of individuals, who during life had their hearing recorded with the audiometer. In some of these patients marked loss of hearing was recorded in certain parts of the tone scale. In comparing the histologic findings and the loss in hearing, it was found that areas of degeneration in the cochlea and associated nerve elements were found to be present, and the extent and location of these diseased areas coincided with fair accuracy to the position on the audiometer reading in which losses of hearing were recorded. This and the work of Wittmaack,[3] who produced degenerative areas in the cochlea of animals by subjecting them to the constant noises of a definite pitch, confirms the idea that the basal portion of the cochlea conveys the high tones, and the apical portion the low tones, and the intermediate tones are conveyed by the central portions of the cochlea.

Hughson and Crowe[4] have recorded the immediate effects on the hearing by tension and division of the tensor tympani muscle, by disturbing the continuity of the ossicular chain, pressure on and incision of the membrane of the round window. No record has as yet appeared regarding effects on hearing of the experimental animal after these procedures have had time to heal and scar tissue to form. Cases of chronic suppuration of the middle

ear with destruction of one or more components of the ossicular chain and most of the drum are very common. Sharp drops in hearing for the low tones are rarely recorded, while occasionally the tones of the upper register are markedly impaired. A large number of cases show hearing losses of equal severity throughout the tone scale. Figure 1 is the hearing record of a female, aged 18 years. Since infancy she had a purulent discharge from her right ear. Lately, she developed vertigo and headaches. Radiogram of petrous portion of the temporal bone revealed an area of destruction at the tip. Radical mastoidectomy revealed a fistula leading into the petrous bone. She healed up completely; the discharge stopped. The audiogram of February 20, 1933, shows a definite improvement in hearing. At the time of operation the remains of the drum, malleus, incus, and the tendon of the tensor tympani muscle were removed. It is evident from this that some compensatory or regulatory factor steps in to take the place of the finely balanced mechanism which exists in the middle ear during health.

FIGURE 1.

Audiogram of Miss R. G. Chronic suppuration of petrous pyramid, right side.



Right ear.

Percentage loss..	66	45	45	44	38	27	43	43—Oct. 28, 1932.
Percentage loss..	41	28	32	28	23	12	8	38—Feb. 20, 1933.

October 28, 1932. ———— February 20, 1933. — — — — —

December 6, 1932. Radical mastoid operation and drainage of petrosa. Recovery.

There is very little factual basis for our understanding as to how the sound gains access to the cochlea and in what manner the cochlea translates auditory stimuli into nerve impulses. There is no evidence to show that the sound is transmitted into the auditory labyrinth by means of the ossicular chain or the round window. It is likely that either of these routes may be the avenue by which auditory waves gain entrance to the cochlea. The auditory theory of Helmholtz is that tones cause a vibration of the basilar membrane and that the basilar membrane has the property to vibrate in its various portions resonantly with the tones composing the sound. These vibrations in turn stimulate the corresponding hair cells in the organ of Corti. From this it may be understood that Helmholtz believed the basal membrane to be similar to a harp or piano in which the individual fibers represent a tone. The idea is not generally accepted. Wegel[5] says: "All discrimination as to pitch and intensity is a function exclusively of the properties of the auditory tract.

"In the normal ear the continued vibrations at threshold value probably induce in the tissue a metabolic or extra-resting phase.

"The diffuse electrode chronaxie of the auditory nerve trunk is probably about 0.0001 seconds.

"When vibration takes place there is in general a motion of the organ of Corti normal to the plane of the basilar membrane and a synchronized longitudinal motion of the fluid of scala media. The result of these motions is to apply a shearing stress on cilia of the hair cells and a consequent extensional strain on some excitable interface on or near the cilia."

It is suggested in the article by Wegel that as a result of this stimulus on the cilia and subjoined cell structure a metabolic change occurs which transforms the vibration into electrical current. This current in turn is conveyed to the nerve fibers in contact with or adjacent to the hair cell and acts as the nerve impulse which carries the auditory stimulus to the brain. The metabolic phase (latent period or chronaxie), according to him, may be susceptible to measurement.

Wever and Bray[6] placed an electrode on the exposed auditory nerve of the cat and grounded it in an indifferent part of the body. This was connected with an amplifier and telephones.

Sounds produced at the external ear of the cat were faithfully reproduced on the telephones. They conclude that the electrical changes which produce the sound arise in the auditory nerve and are not due to mechanical or electric effects in the cochlea.

Adrian[7] repeated the work of Wever and Bray and was able to pick up sounds by placing an electrode on the labyrinthine capsule, and particularly the round window, indicating that the electric currents produced as the result of sound stimulus arise in the cochlea. He contends that if Wever and Bray are correct it must be predicated on the assumption that nerve fibers have an extremely short refractory period for the conversion of auditory stimuli into nerve impulses.

HEARING ACUITY.

It is generally accepted that by kindness of nature some people are born with better bodies than others. The individual whose muscle co-ordination, digestion, sight, hearing, smell, taste, balance and mental equipment function better than the average may be classed in the category of supermen and women. Such persons finally excel in athletics, in the professions and business. That it is possible for an individual to be born with hearing above the average while the other senses and organs do not rise to that level is an oft repeated observation. On the other hand, some people are endowed with subnormal hearing, while the great majority have average good aural acuity.

It is presumed that if an individual goes through life without having experienced any of the major illnesses, sustained no injuries, was not exposed to severe weather changes and suffered no ear infections, his hearing would remain sufficiently acute for all practical purposes through a long life. There are few people who enjoy this freedom from diseases or injury, and so we find that the hearing organ becomes involved in some form of degenerative process as age advances. It is common knowledge that in those people who are in apparently good health hearing depreciation is not noticed until some time after the sixtieth year of life. A noticeable drop in hearing capacity below the age of 60 and even after this time should be considered as evidence that

there is something wrong with the health of that person, because good hearing has been repeatedly observed in people over 60 years of age. We do not believe that the age of an individual should be gauged by time. People of 70 years of age are encountered who appear as young as people of fifty years. It does seem, therefore, that the hearing acuity of one person at a given age cannot be compared with that of other persons of a similar age for the purpose of coming to a conclusion as to its normalcy.

Bunch[8] made a statistical inquiry for the purpose of establishing a means by which a given audiometric reading of a person in a certain decade of life may be evaluated as to its probable abnormality. Patients in the medical and surgical wards who gave no history of impaired hearing had their hearing tested with the 1A audiometer. There were sixty-eight cases in the third decade, seventy in the fourth, seventy-eight in the fifth, eighty-five in the sixth and fifty-two in the seventh. The number was doubled in each instance by using the audiograms from both ears. Scattergrams were made of the records for each decade, and the mean and mean variations estimated and plotted. From the standpoint of large experience in reading audiograms of the deaf and those with apparently normal hearing in all age groups, it appears to the essayist that even the upper mean variation in the charts for the groups up to and including the fifth decade, is below that commonly encountered in people in good health and who do not complain of hearing defect or have not had inflammatory diseases or injuries to the hearing mechanism. This especially applies to lowered hearing acuity for the low tones. The upper mean variation for people in the sixth decade more nearly approximates expectancy.

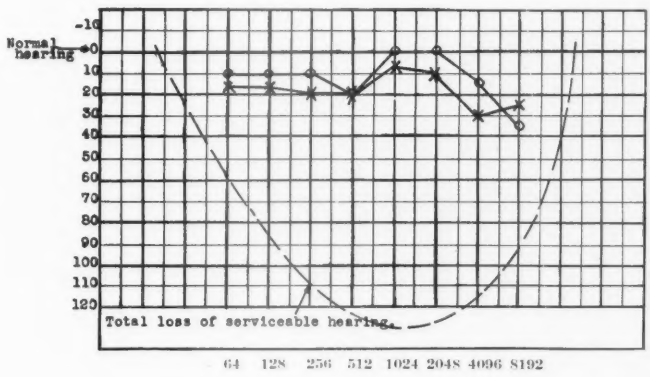
In studying the scattergrams one cannot fail to be impressed with the number and extent of the threshold depreciations for the 4096 frequency in the patients in the groups above the third decade. This dip at 4096 was described by Drury,[9] in cases of syphilis. He also referred to this finding in connection with his observations associated with liver disorders. Ciocco[10] calls our attention attention to greater hearing loss at 4096 and regards it "as a first indication of a degenerative process which results in the loss of acuity for all high tones." The work of Bunch is

based on audiometric study of patients from the medical and surgical wards of the hospital. The results obtained are what would be expected. The hearing organ is the most recent acquisition of man in his march through the ages. It is extremely sensitive to injury from nutritional and toxic disorders. It is here submitted that the only way to determine auditory acuity at different ages is to base the investigation not on calendar years but on health years. This can be accomplished by establishing certain standards of health age by studying the vital capacity of each individual and grouping the cases accordingly. In this way a reliable yardstick will be available by which one may determine if a given audiometric record comes within the range of normal.

Appreciation of slight loss in hearing is dependent upon the intelligence and environment of the individual. The day laborer who works in noisy surroundings and in whose home everyone talks loudly is not liable to complain of hearing loss as early as an individual whose surroundings are quiet. Some people appeal for help because when in groups the conversation is lost to them. This is the earliest manifestation of a beginning deafness that I have encountered. Fig. 2 is a hearing record of a patient who consulted me after realizing the fact that he was lost in a group carrying on a lively conversation.

FIGURE 2.

Audiogram of Mr. X. Age 47 years.



The hearing loss is more severe with those who consult the otologist because they do not hear all the words over the telephone. Fig. 3 is an audiogram of such a patient.

FIGURE 3.

Audiogram of Miss A. G. Age 16 years.

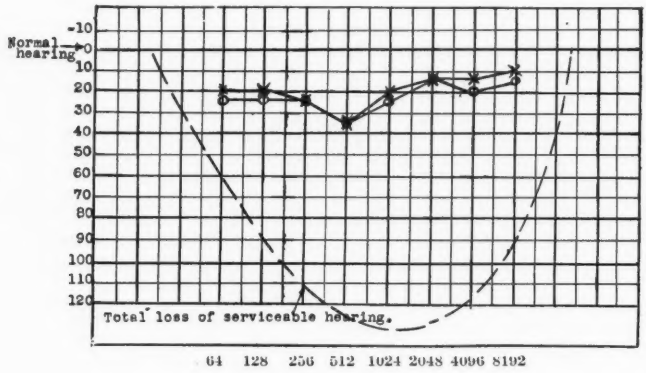
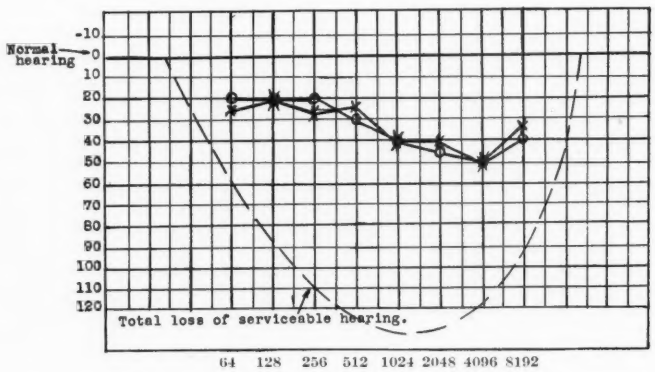


FIGURE 4.

Audiogram of Mr. F. M. Age 43 years.



Musicians complain of hearing loss when the sounds are distorted or they are unable to tune their instruments, particularly in the higher tones, with accuracy. Fig. 4 is an audiogram of a patient whose first knowledge of approaching deafness occurred

when his wife called his attention to the fact that he did not properly tune the high notes of her harp.

When the hearing loss is more severe the patient does not hear the buzzer, door or telephone bell.

BONE CONDUCTION.

It is the ability to hear a note or a noise when the activated source of these is placed upon the head. In testing for bone conduction, the watch, acoumeter and tuning fork are used. Tuning forks of very low frequency are not satisfactory because they not only convey sound but also vibrations which may be interpreted by the subject as sound. Forks of high pitch have the disadvantage that they also may be heard by air when the base of the fork is placed too near the ear. In this case when the fork is placed on the mastoid the vibrating prongs may be tilted away from the ear. If the patient has good hearing with that ear he may hear the fork by air conduction, notwithstanding this precaution. For this reason the 128 D. V. and 256 D. V. forks are the most useful. The normal ear hears bone conduction one-half the length of time as through air conduction with the 128 D. V. fork. In cases of disease of the middle ear the air conduction time is shortened, while the bone conduction is either unaffected or slightly shortened. In some cases of deafness bone conduction is markedly prolonged while hearing by air is reduced.

Theory.—There has been a great deal of speculation as to how the vibrations of a tuning fork placed on any part of the skull reach the cochlea and are appreciated as sound. As the result of this, three hypotheses have been put forth to explain this phenomenon. (1) Craniolabyrinthine—According to its proponents, the activating force causes the skull to vibrate, and these vibrations are conveyed to the cochlea directly through the bony capsule of the labyrinth. This idea has been generally discarded because it is felt that the labyrinthine capsule has no special capacity for the conveyance of sound. (2) Craniotympanic—Here it is supposed that the vibrations reach the tympanic ring; this, in turn, sets the ear drum in motion, which motion is propagated in the usual way to the ossicles, thence to the cochlea. This route is the one that is generally accepted, but is difficult to reconcile

in view of the fact that bone conduction is better heard than air conduction when there is disease of the middle ear with destruction of the drum, if the labyrinth is not evidently involved. (3) Craniopneumotympanic—This is the idea evolved by Becker.[11] According to him, the vibrations of the tuning fork when placed on the head are conveyed to all parts of the skull and set in motion the air contained in cavities. The larger the air containing cavity the greater the amount of vibration that is produced (resonance). The air in the external auditory canal is also caused to vibrate and conveys these vibrations to the drum; then the sound is carried to the cochlea in the same manner as through air. If the finger is inserted into the ear, the sound of the vibrating tuning fork is increased. When the palm of the hand is placed over the ear the tuning fork is heard with greater intensity than when the finger is inserted into the ear. This is explained on the basis that the capacity of the external auditory canal as a resonating chamber is larger when the palm of the hand is placed over the external ear than when the finger is inserted into the external auditory meatus. Becker calls bone conduction a modified form of air conduction.

Politzer[12] says that bone conduction is heard better in the ear that has disease of the external auditory canal and middle ear, if the labyrinth is not involved. It is not of value in chronic bilateral affections of the middle ear. In some instances, where bone conduction is found prolonged with the low frequency forks, the bone conduction with the higher pitched forks have shortened bone conduction.

Ciocco[13] states because of certain observations on the acuity of hearing in cases of disease of the middle ear "we must conclude that middle ear lesions do in some degree affect acuity for high tones." He believes that middle ear infections can produce degeneration of the inner ear. Also that "the membrana fenestræ rotundæ is semipermeable, and that its thickening and scarring can be considered a protection of nature to safeguard the internal ear."

Hughson and Crowe, speaking in reference to the effect of tension and division of the tensor tympani muscle (tension impairs transmission for all low tones and division impairs transmis-

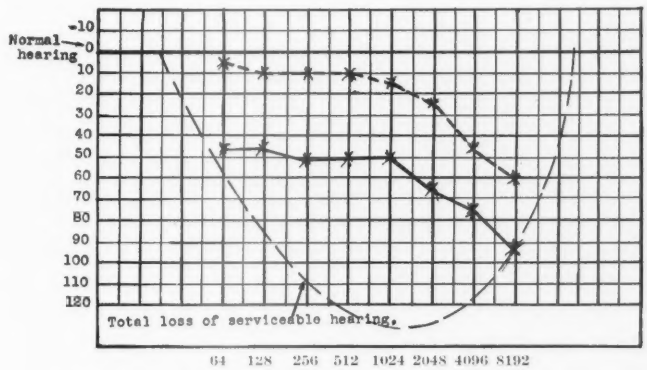
sion of high tones), say: "If this proves to be the case, we will have to modify one of the fundamental otologic concepts that 'the loss of high tones is always due to inner ear lesions and cannot be restored by treatment.'"

For the past two and one-half years I have kept hearing records of patients with acute and chronic otitis media, and can easily subscribe to the observations and deductions of Dr. Ciocco. The majority of my cases show a greater hearing loss for the high tones than the lower frequencies. The high tones are usually depressed in cases of acute and chronic purulent inflammation of the middle ear. In the acute cases the hearing for high tones is immediately depressed, so much so that the ability to hear 8192 D. V., and even in some cases 4096 D. V., is lost, and in a few cases it is not restored when the inflammation subsides. Succinctly stated, the loss of acuity for high tones following middle ear disease may be due to atrophy or relaxation of the tensor tympani muscle or involvement of a portion of the basal turns of the cochlea by extension of the inflammatory process through the round window.

Fig. 5 illustrates the drop in hearing of a patient suffering with acute otitis media and also a record of the same ear during convalescence.

FIGURE 5.

Audiogram of Mr. C. J. Left ear.

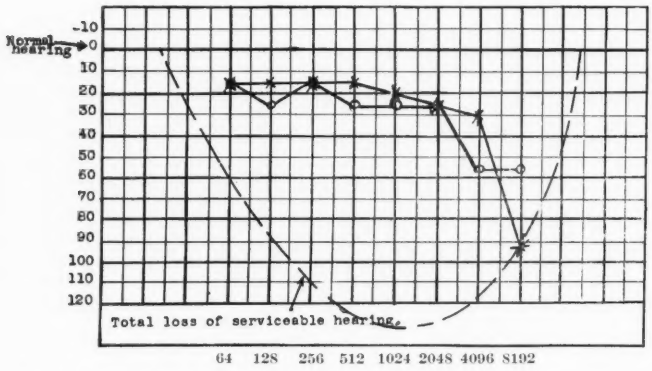


———— July 11, 1931. Acute purulent otitis media.
— — — — — July 22, 1931. During convalescence.

Fig. 6 is the audiogram of a patient who has scarring and displacement of the drums as the result of a previous bilateral, chronic, purulent otitis media.

FIGURE 6.

Audiogram of Mrs. E. S. Age 32 years.



In view of the above and other observations, it is necessary to re-evaluate the significance and the theory of the phenomenon of bone conduction. Certainly, the idea that disease of the middle ear, and especially the inner wall of the middle ear, produces certain changes in the resonance of the bony capsule of the cochlea so that the low tones are better heard by bone conduction than air conduction, is subject to serious doubt.

Gray[14] finds from his pathologic studies in otosclerosis that the nerve elements are primarily involved and the bony lesion occurs later. He finds that early in the progress of the disease the medullary sheaths and neurilemma disappear, leaving the axis cylinders free. Later the axis cylinders undergo atrophy. Dr. Gray explains paracusis Willisii on the basis that as a result of other sounds occurring during a conversation between one person and a sufferer with otosclerosis, the incidental sounds gain access to the nerve fibers conveying the conversation and amplify the intensity of the conversational sound by augmenting the number of nerve impulses that are carried by these denuded axis cylinders per unit of time. The axis cylinders in the cochlear nerve of the patient suffering with otosclerosis have lost their insulation and

the nerve impulses are conveyed from one to the other when these nerve fibers are in contact with each other. In this connection, Dr. Gray says as follows: "In the normal individual the nerve currents in the cochlear nerve created by the conversation derive no increase of intensity from the nerve currents produced by the surrounding noise, whereas in the case of the paracusic individual they do. Consequently, the latter hears relatively better in a noise than a normal individual. Furthermore, it is quite possible that the paracusic may hear absolutely better in a noise than in silent surroundings."

Mr. Somerville Hastings[15] observed that when a patient suffering with otosclerosis and having paracusis as one of the symptoms has a vibrating fork applied to one mastoid and a noise entering the opposite ear, the bone conduction intensity in the other ear is definitely augmented. Dr. Gray calls this phenomena crossed paracusis and explains the method of its occurrence on the hypothesis that not only are the pathways between the cochlea and the medulla involved in a process of degeneration, but also the nerve tracts which lead from the medulla to the auditory center in the temporal lobe. The nerve fibers from the cochlea decussate in the medulla so that impulses from one ear go to the temporal lobe of the opposite side, but a smaller number go to the temporal lobe of the same side. Dr. Gray suggests that the noise augments the sound of the tuning fork by it gaining access to the channels which are conveying the sound, thus increasing the intensity of the nerve impulses. According to him, this can be produced if adjoining axis cylinders in the afferent pathways are denuded of their insulation.

Knudsen and Jones[16] have reported that prolonged bone conduction occurs in the presence of noise, whereas, in the soundproof room prolonged bone conduction is rarely observed.

Leaving out the variations in bone conduction which are observed in cases of middle ear disease which are never prolonged above that which is found in normal individuals, I consider that actual prolongation of bone conduction is a manifestation of disease of the nerve elements in the cochlea and can be considered to be a modification of paracusis Willisii. There is with every noise a vibration. With the higher tones, the vibration is not

appreciated because the sound waves are shorter and more rapid in their occurrence. The vibration which occurs with the lower tones is perceptible to the individual, and as the tone is lowered the vibration becomes more apparent and the tone inaudible. I suggest that the occurrence of paracusis and the prolongation of bone conduction is not dependent upon extraneous sounds but is due to the effect of vibrations accompanying these sounds. Paracusis Willisii, prolonged bone conduction and the phenomenon described by Mr. Hastings can be easily explained on the basis that the incidental vibrations jostle together and make a larger contact of the noninsulated axis cylinders in cases of otosclerosis and thus afford a larger nerve surface for the conveyance of the spoken voice and tones when such are produced in the presence of vibrations associated with extraneous sounds.

Crowe,[17] in answer to a question initiated by his presentation before the meeting of the American Otological Society, said as follows: "In the cat, the bulla and the middle ear is opened through an incision in the neck; air conduction and bone conduction are tested and charted as described in previous publications. The middle ear and bulla is then filled with mercury. This fixes both the stapes and round window membrane. Under these conditions air conduction is nil, but bone conduction is actually and measurably improved."

The artificial condition produced in the above experiment does not approach the pathologic conditions which arise in the middle ear from all causes. The molecular tension and lack of capillary attraction of mercury is far from being comparable to that of exudates, fibrous tissue, tissue necrosis or even new bone formation. It therefore cannot be used as a basis for a deduction that abnormalities in bone conduction are due to fixation of the stapes or the membrane of the round window.

SUMMARY.

1. The elements which enter into our understanding of the physiology of hearing are recorded and discussed.

2. Various phases and problems of hearing acuity are present. ed. A method for determining the normal hearing at different ages is offered.

3. The theories of bone conduction are reviewed. A plausible theory for the phenomenon of prolonged bone conduction is presented.

The text is illustrated with descriptive audiograms.

2 WEST 87TH STREET.

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LXIII.

MALIGNANCIES OF THE UPPER AIR PASSAGES: A STATISTICAL REVIEW.*

SAMUEL SALINGER, M. D.,

CHICAGO.

This paper is an attempt to summarize the results of treatment of malignancy of the upper air passages as revealed in a series of publications in the German literature and to compare them with similar statistics from other large centers throughout the world. Particular emphasis will be laid on the X-ray and radium treatment of the inoperable cases which comprise such a large percentage of the total. Despite long continued and repeated failure in the use of these measures over a period of years the work has gone on unceasingly, each new detail of technic advanced being carefully studied and each new method obtaining a modicum of success being gratefully received and critically studied. Certainly we have not reached the end of our hopes nor have we by any means exhausted the possibilities of radiation. Definite impetus has been given the work by advances along three lines: first, earlier diagnosis through better morphologic studies; second, increasing appreciation of the degrees of radio sensitivity of various types of malignancy; and third, the new technic of radiation as advanced by Coutard and Berven.

We find observers all over the world giving particular attention to the radio sensitivity of the particular growth in question and applying the principles of teleradium and protracted fractional radiation more intelligently and with greater confidence. Broder's classification of carcinoma has offered us an excellent guide for determining the degree of malignancy, and more recently a number of authors have taken to a classification based on the radio sensitivity of the growth which, broadly speaking, falls into three groups: (1) Lympho-epithelioma, (2) transitional cell carcinoma,

*Read before the Middle Section of the Laryngological, Rhinological and Otological Society, Cincinnati, Ohio, January 9, 1933.

and (3) squamous cell carcinoma. Those in group one are most sensitive to radiation and those in group three least sensitive.

There are numerous subdivisions of these groups exhibiting gradations of sensitivity according to the type and distribution of their cellular elements. Naturally, if the type of tumor is carefully studied from this point of view the degree of radiation can be more accurately planned and the prognosis more definitely established. The subject of radio sensitivity has been gone into rather fully both here and abroad, and the information is by now fairly well distributed. We shall therefore pass on to a consideration of some of the views expressed abroad on the cellular reaction to radiation both in the tumor itself and in the normal tissues exposed.

Engelman[4] made a very exhaustive study of tissue changes resulting from radiation through biopsies taken at regular intervals during the course of treatment. He studied the effects on various types of growth under the single lethal dose, the repeated smaller doses and the newer protracted fractional radiation of Coutard, and comes to the conclusion that there is no "specific morphologic action of X-rays or radium on tumor cells." The cell degeneration observed was of the same type long known in Pathologic Anatomic from other forms of cell injury. With regard to the effect on normal tissues he found these less disturbing under protracted radiation than the formerly employed technic. Tissues remaining unchanged are the cellular elements of the blood, the sweat and sebaceous glands, the surface epithelium (skin and mucosa). Atrophic changes were observed in the salivary glands and the connective tissue with its vascular apparatus. More marked atrophy was usually noted in the lymphatic structures of the throat, although such sections as remained unaffected continued to function normally. Properly supervised radiation caused no changes in bone or cartilage. In connection with the last named it is interesting to note the measures taken by various authors to prevent necrosis of laryngeal cartilage exposed to radium.

For instance, Seiffert[19] and Hegener[7] advise making a window in the thyroid cartilage by removing a segment and placing the

radium placque in direct contact with the invaded laryngeal tissue. Seiffert, quoting Ledoux, also mentions the insertion of a radium needle directly into a circumscribed cordal lesion through a small buttonhole opening through the thyroid cartilage. He claims 200 to 300 mg. hours sufficient in such a case to cure the lesion. In the window cases the dose employed was 1,500 mg. hours. Halberstadter[5] used a radium placque without removal of cartilage in cases where the latter was already invaded. He employed a daily dose of 75 to 150 mg. hours up to a maximum of 1,000 to 12,000 mg. hours, using 1 mm. of platinum for filtration. Of eleven cases thus treated there were two with late necrosis. Harmer[6] used the Ledoux technic of removal of a cartilage before radiation. Thielman[21] sewed a radium placque containing 15 to 20 mg. under the perichondrium without removing the cartilage, leaving it in place for seven days. The dosage was up to 1,800 mg. hours. He reports only one case of cartilage necrosis in a series of five.

Supporting Engelman's findings, the results under the protracted fractional method of X-rays and the teleradium as reported by other investigators reveal few instances of cartilage necrosis. Cutler and I gave 80,000 mg. hours in divided doses over four fields with the radium bomb in a case of carcinoma of the larynx with only a transient swelling of the perichondrium.

A general survey of the reports from the German clinics discloses a diversity of methods of radiation with varying degrees of disappointment in the results attained. Most of the authors have in the past two years given more attention to Coutard's method of prolonged fractional radiation and express satisfaction in the limited number of cases thus far treated in this manner. One objection to the Coutard method advanced by Hunerman[10] is that the cost is prohibitive for clinic patients. He quotes Zwerg's estimate that an average treatment involving 6,000 r. in divided doses would use up to one-tenth the guaranteed life of a Mueller tube costing 1,800 marks. Thus the actual cost of the treatment would be at least 180 marks. Also, inasmuch as the treatment must be extended over a period of thirty days, during which time the patient is confined to the hospital, he estimates the total cost for hospitalization, nursing, X-rays, etc., to amount to

at least 1,150 marks. In the main, however, the Coutard method has been accepted with much hope and enthusiasm and wherever the circumstances would permit has been employed. Naturally the number of cases thus treated has been meager and the time elapsed too short to enable one to draw definite conclusions. The progress of the work in these clinics will undoubtedly be followed with great interest, and the results within the next two years may be fraught with far reaching possibilities.

Before discussing the details of the accompanying tables it would not be amiss to point out the essential facts in connection with the Coutard method of protracted fractional X-rays and the teleradium of Berven. In both methods the agent is placed at a much greater distance from the skin than in the older methods: For X-rays the tube distance is between 60 and 100 cm.; for radium the distance of the bomb is 6 cm. Secondly, the individual dose is much less than that previously used, but is repeated at daily or longer intervals over a period of time, reaching the affected area through different areas until a total radiation many times the normal skin erythema dose has been attained. It is thus possible to give from 6,000 to 15,000 r., or 10 to 20 H. E. D., without permanent damage to the overlying structures. The criterion of adequate dosage has been described as an "epidermite" or "epithelite." This is a reaction which comes on about the twelfth to the fourteenth day from the onset of the treatment: the skin becomes red and the patient experiences pricking sensations in an area about 10 cm. square. The hairs fall out and about the middle of the fourth week the skin becomes a deep red and finally grayish until the superficial layers slough off. The ulceration which remains usually healed in the seventh week, restoring the skin a little duskier and drier than normal, but otherwise intact in all its layers. The important point about this reaction is its being confined to the superficial layers of the skin in contrast to the X-ray burns previously observed under the older technic which always involved the entire dermal structure and left long standing deep indolent ulcers.

The reaction in the mucosa, as described by Berven,[1] begins about the fourteenth day with slight dysphagia and dryness of the

mucosa. Soon the latter is coated with grayish white spots becoming confluent until a false membrane is formed. This may cover the tonsils, palate and pharynx and lasts from seven to ten days. It gradually disappears, leaving the mucosa reddened but intact. Berven claims that the skin reaction from teleradium is much less severe than that resulting from X-rays.

EPIPHARYNX.

Coming now to a consideration of the results by groups according to location, we find that malignancies of the epipharynx have yielded very poor results up to recently. Most cases in this category are first seen when far advanced, frequently with extensive metastases and therefore less favorable for treatment. Inasmuch as the primary lesion is nearly always inoperable, treatment has been confined almost solely to the use of X-ray and radium.

The accompanying tables show very few cures by the older methods except in the sarcoma cases, which are more amenable to radiation than carcinoma. For instance, Jung and Martenstein,[11] Hunerman,[10] Richter,[17] Luscher[13] and Hesse[8] fail to show a single recovery in carcinoma of the epipharynx treated by the older methods. Zuppinger,[23] whose series is larger than any of the above named authors, claims 12 per cent of recoveries in a group of thirty-two treated by surgery alone or combined with X-rays. Harmer,[6] by combining X-rays with local radium applications totaling 1,500 to 3,000 mg. hours, obtained 11 per cent cures in eighty-six carcinoma and 32 per cent in sixty-three sarcoma for periods up to five years.

On the other hand, since employing the Coutard method some authors are showing astounding primary results. For instance, Kraus[12] has healed eight out of ten cases (80 per cent) by protracted fractional X-rays and advises against removal of the glands. Naturally these cases have not been observed long enough (one year) for definite conclusions to be available. The brilliant primary results, however, are sufficiently startling to merit respect. Schintz[18] and Richter[17] also in the past year have attained equally good results in the few cases thus treated. Certainly, one must admit that progress is being made.

SINUSES AND MAXILLA.

Results in this group are more difficult to evaluate because of the diversity of methods employed in addition to radiation. In most clinics malignancy of the sinuses and upper jaw are considered operable and are so treated. Either radical surgery or surgery plus electrocoagulation are generally employed, followed by radium or X-rays. The tendency seems to be in the direction of more prolonged radiation following the surgical procedure than formerly used. It is interesting to note that despite the variety of procedure statistics as to results run fairly uniform. The percentages are higher in the sarcoma than the carcinoma cases and higher in the series where radical surgery was combined with radiation. The number of cases reported from the German clinics is smaller than those from England and America, but the percentage of recoveries compared favorably. Whether any advances in this sphere may be expected is difficult to foretell. At the present time surgery seems to hold the foreground with radiation playing a secondary but important rôle.

HYPOPHARYNX.

It is rather difficult to compare statistics of cases in this group because of the variety of locations and types of growth encountered. Some of the authors include malignancy of the tongue, pyriform sinus, pharyngeal walls and postcricoid region within one group, while others note their results separately for each location. However, all malignancies in the areas mentioned have certain characteristics in common which offer a basis for comparing similar methods of treatment, namely, they are in the main inoperable and they are accompanied by early glandular metastases. Here, as in malignancy of the epipharynx, two facts stand out prominently: first, that the sarcomas are much more amenable to radiation than the carcinomas; and second, the results under the newer methods of Coutard are far better than the older technic.

Interstitial radiation by radium needles and implants has given way to surface application or the radium bomb, and the single large or repeated dose of X-rays is being supplanted by the

protracted fractional X-rays already mentioned. Some of the older series, such as those of Jung and Martenstein,[11] Luscher[13] and Hesse[8] gave practically no recoveries in the carcinoma cases. Even the larger series of Harmer[6] and Quick,[16] which run into hundreds, yield only 5 per cent to 10 per cent of recoveries for periods of one to ten years. Coutard's[2] results under the newer method are somewhat better. He shows 20 per cent recoveries up to two years; after that point the percentage falls rapidly.

Stewart Harrison[20] reports from the Zurich clinic a series of eighty-four cases under treatment for the past three years by the Coutard method, with primary recoveries in 25 per cent to 100 per cent, depending on the location of the growth. He divides his cases into numerous groups according to original location of the lesion and records not only the particular morphology in each case but also gives the exact amount of radiation employed. For instance, carcinoma of the epidermoid type involving the base of the tongue, vallecula, or aryepiglottic folds received from 6,360 r. to 8,000 r. over three to four skin fields as against only 3,450 r. to 5,300 r. in the case of a similar type of carcinoma located in the pyriform sinus. His patients have been symptom free for periods of one and one-half to three years, and the large percentage thus healed is astounding in view of the failures recorded in the older series.

TONSILS.

Malignancy of the tonsil deserves separate consideration because it is one location in which the prognosis has been changed from the hopeless to the definitely favorable. One need only compare Berven's[1] records for the periods 1919-1924 with those of 1923-1927 to realize that a remarkable advance has been made. Under the old radium technic he treated twenty-eight cases with 10 per cent recoveries for one year and no cures beyond that period. With the newer teleradium his series of eighteen, 50 per cent were healed one year; 44 per cent, two years, and 39 per cent, three years. The contrast is striking and the figures speak for themselves. Coutard himself could report no cures under the older method of X-ray, but with the protracted fractional technic he obtained 27 per cent of cures for three years and 19 per cent

for five years. Naturally the prognosis is better in the radio sensitive than the radio resistant carcinomas.

LARYNX.

Under this heading we find a variety of statistics which are often not comparable because of the diversity of treatment. Nearly all the authors still yield to surgery as first choice in the strictly operable cases, but in the borderline and extrinsic group there is a divergence of therapeutic measures. There is a tendency to avoid radical surgery where the prospect of complete eradication of the growth is doubtful and to rely on intensive radiation. The methods employed have already been mentioned in an earlier paragraph. Some of the authors while enthusiastically endorsing the Coutard and Berven technic are still intrigued by the possibilities of direct radiation through fenestration of the thyroid. The fear expressed that the large doses necessary to reach the laryngeal mucosa must necessarily endanger the overlying skin as well as the laryngeal cartilages, does not, however, seem to be borne out in the reports of Hunerman, Luscher, Schintz, Coutard and Stewart Harrison. Here again, as in the case of the tonsil and hypopharynx, they show percentages of recovery all the way from 20 per cent to 63 per cent for the inoperable carcinomas of the larynx. Naturally we may expect recurrences in a large number of this group as time goes on, but even discounting this factor, we feel confident that a five year survey will reveal a definite proportion of cured cases in a class that formerly offered no hope.

Finally there is this to be said: Although the survey here presented is necessarily sketchy, because of the wide field covered, nevertheless, if taken in conjunction with a study of the accompanying tables it must lead to the conclusion that carcinoma of the upper passages is emerging from the dreary past to a more hopeful future. The technic of Coutard, Berven, Hegener and their co-workers is very diligently studied and employed wherever the physical means are at hand, and we may confidently expect within the next five years to see these methods vindicated in still higher percentages of recoveries.

25 East Washington Street.

EPIPHARYNX.

Clinic and Author	Number of Cases	Number Healed	Per Cent	Duration	Method
Hunerman (Dusseldorf)	2 Ca. 1 Sa.	0	0		Protracted fractional X-rays. Cases well advanced with metastases.
Richter (Erlanger)	4 Ca. 2 Sa. (1919-26)	0 1	0 50%	5 years	Older method of X-rays. One case of carcinoma treated 1930-31 by newer repeated X-rays; primary healing.
Luscher (Bern)	6 Ca.	0	0		Local radium 800-3,600 mg. hours. All cases had metastases.
	1 (lympho-epithel.)	1	100%	5 years	Harpoons of radium 3.3 mg. for 48½ hours; also contact capsules 10-20 mg. (1-2 mm. platinum). Glands excised.
Hesse (Koenigsberg)	3	0	0		1—Radium 1,200 mg. hours. 1—Incomplete Coutard. 1—3 Exposures full X-rays.
Schintz (Zurich)	4 (2 cases completed)	2	100%	Not stated (Recent)	Coutard.
Jung and Martenstein (Breslau)	10	0	0		Older methods of X-rays.
	12	3	25%	1½ to 2 years	Combined X-rays (protracted fractional) and local radium.
Kraus (Prague)	10	8	80%	Less than 1 year	Protracted fractional X-rays. Same for glands. Does not advise excision.
Zuppinger	32	4	12%		Surgery alone or combined with X-rays.
Harmer (England)	86 Ca. 63 Sa.	10 22	11% 32%	2 years 2 to 5 yrs.	Mostly highly malignant and inoperable cases.
Hegener (Hamburg)	9	3	33%	15 to 18 months	Interstitial radium; surface radiation; and Coutard X-rays.

SINUSES AND SUPERIOR MAXILLA.

Clinic and Author	Number of Cases	Number Healed	Per Cent	Duration	Method
Hunerman (Dusseldorf)	1 Sa. Ethmoid	1	100%	1 year	Electro-coagulation and protracted X-rays.
	1 Ca. Maxilla	0	0		Electro-coagulation and 975 mg. hours radium and X-ray.
Richter (Erlanger)	16 { 12 Ca., 4 Sa., 1 L. S.	6	40%		Surgery plus X-rays. (Likes the Coutard method and using it lately, but not included in this series.)
	8 { 4 Ca., 4 Sa.	2	25%		X-rays alone.
Luscher (Bern)	22 (15 inoperable) (7 metastases)	4	18%	1½ to 3½ years	Radical surgery or coagulation followed by radium (700-5,000 mg. hours). Later cases had more intensive radiation through moulage for total 10,000-14,000 mg. hours.
Hesse (Koenigsberg)	26	8	31%		Radical surgery and radium and intensive X-rays.
Thielman (Bonn)	Statistics Not Given				Electro-coagulation followed by radium: single large dose. Removal of gland after raying.
Harmer (England)	53	10	20%	More than 1 year	Surgery plus radium and X-rays.
New (Rochester)	70—Primary in antrum		30%	1 to 8 yrs.	Surgery electro-coagulation plus X-rays and radium.
	27—Secondary in antrum		51%	1 to 8 yrs.	
Quick (New York)	103—Antrum	16	15%	1 to 10 yrs.	Surgery and radium.
	122—Maxilla	29	23%	1 to 10 yrs.	
Hegener (Hamburg)	11	1	9%	More than 6 months	Surgery; radium; Coutard X-rays.

HYPOPHARYNX.

Clinic and Author	Number of Cases	Number Healed	Per Cent	Duration	Method
Hunerman (Dusseldorf)	6	1	18%	More than 6 months	Fractional X-rays.
Luscher (Bern)	7	1	15%	Very short	800-2,000 mg. hours radium by Oesophageal carrier. 1.25 mm. platinum filter. All advanced cases; 4 with glands; 3 inoperable.
Jung and Martenstein (Breslau)	18	0	0		Four treated by X-rays and radium; one greatly improved for six months. Two others observed two years. Series included extrinsic carcinoma of larynx.
Hesse (Koenigsberg)	12	3 (all Sa.)	25%	1 to 1½ years	Radium (500 mg. hours and two full doses of X-rays). Electro-coagulation plus X-rays alone or with radium.
Thielman (Bonn)		No Statistics Quoted			Radium carrier fastened to teeth left in place 24 hours. Palliative results only.
Harmer (England)	106	11	10%	1 to 5 years	X-rays.
Coutard			20%	2 years	After two years the percentage of cured cases falls rapidly.
Quick (New York)	221	9	5%	1 to 10 years	Radium.
Stewart Harrison (Zurich)	84		25% to 100% depending on location	½ to 2½ years	Coutard. Series included base of tongue, vallecula, mesopharynx, epiglottis, folds, pyriform sinus, postcricoid and hypopharynx.
Hegener (Hamburg)	26 (1929-1931)	4	16%	18 months or more	Coutard.

TONSILS.

Clinic and Author	Number of Cases	Number Healed	Per Cent	Duration	Method
Richter (Erlanger)	7 Ca. 1 Sa.	1 1	14% 100%		X-rays. Surgery and X-rays.
Luscher (Bern)	14	0	0		Eleven with metastases; 7 inoperable treated with radium harpoons 1,000-6,000 mg. hours.
Jung and Martenstein (Breslau)	18 10	2 (Sa.) 3 (Sa.) (Sa.) (Ca.)	11% 33%	More than 6 months 6 months 6 months 1 year	X-rays only after surgery. X-rays and radium.
Stewart Harrison (Zurich)	24 (19 completed)	9	48%	½ to 2½ years	Coutard. Average of 6,000 r., three fields.
Berven (Stockholm)	28 (1919-1924)		10% 0 0	1 year 2 years 3 years	Old radium technique.
Berveb (Stockholm)	18 (1923-1927)		50% 44% 39%	1 year 2 years 3 years	New method teleradium with radium bomb. Distance of 6 cm. plus filtration; four fields. Daily 2-10 gm. hours. Total about 80 gm. hours.
Coutard	44		27% 19%	3 years 5 years	Protracted fractional X-rays. (Older methods yielded no cures.)
Cutler (Mem. Inst.)	9	9	100%	3 to 11 years	Surgery, X-rays, surface radiation, interstitial radiation.
Hegener (Hamburg)	20 (19 with glands)	11	52%	More than 9 months	Interstitial radium and Coutard X-rays.

LARYNX.

Clinic and Author	Number of Cases	Number Healed	Per Cent	Duration	Method
Hunerman (Dusseldorf)	8 (5 intrinsic)	3	60%	Not stated	Daily doses of ½ H. E. D. to total of 6-12,000 r. 10-20 H. E. D. over 2 or 3 fields lasting 3-4 weeks. Coutard.
Richter (Erlanger)	14 (1919 to 1926)	1	7%	12 cases were inoperable	2 by combined X-rays and radium. 5 by X-rays. 7 by symptomatic.
Luscher (Bern)	50 (Extrinsic)	1	2%	7 years	Lateral pharyngotomy in cured case. All others by radium needles or capsules. A few had X-rays to glands.
Luscher (Bern)	13 (Intrinsic)	4	30%	3 to 5 years	1—Laryngo fissure. 1—Fissure and electrocoagulation. 1—Fissure, radium needles, ext. radium 3,100 mg. hours. 1—Fissure, electrocoagulation.
Hesse (Koenigsberg)	5 (Intrinsic) (16 extensive intralaryngeal)	5 (Cords only) 6 2 0 1	100% 75% 100% 25%	1 to 5 years Not stated	All by thyrotomy: one followed endothermy. 8—Thyrotomy and endothermy. 2—Excision under suspension and endothermy. 1—Sarcoma by X-ray (Coutard). 4—Laryngectomy.
Hesse (Koenigsberg)	(18 extra laryngeal)	4	25%	4 to 5 years	12—Laryngectomy. 3—Protracted fractional X-ray (Coutard). 2—Untreated. 1—Died of Hemorrhage.
Schintz (Zurich)	15 (1929-1932)	(11 cases completed) 7	63%		Coutard method.

Halberstadter (Berlin)	11				Radium without thyroid window. Two cases of late necrosis. Warns of this danger. Advises 1,000 to 12,000 mg. hours with 1 mm. platinum, divided into daily doses of 75-150 mg. hours.
Vogel (Berlin)	14	3	22%	2 to 6 months	Using Coutard method past year. Five additional cases greatly improved. Included 5 base of tongue and 1 postpharyngeal.
Seiffert (Cologne)					Using Coutard method past 18 months. Results not so good. Cases all had glands. Believes X-ray should precede extirpation of glands because the treatment causes them to shrink and often makes inoperable masses operable and leads to better surgery. Finds skin reaction quite constant but mucosa reaction less regular.
Thielman (Bonn)	5	3	60%	½ to 1 year	Radium plaque 15-20 mg. sewed under perichondrium. Cartilage not removed. Left in situ 7 days. Doses up to 1,800 mg. hours. One case of necrosis.
Zuppinger	23 (1918 to 1928)	6	24%		Surgery alone or combined with X-rays.
Coutard	77		32% 21%	3 years 8 years	Protracted fractional X-rays.
Harmer (England) Radium Inst.	8 (Suitable for Thyrotomy)	7	88%	2 to 6 years	Ledoux fenestration of thyroid Interstitial radium and X-ray.
Harmer	20 (Suitable for Laryngectomy)	7	33%	1 for 6 yrs. 1 for 5 yrs. 4 for 2 yrs. 1 for 1 yr.	Pre- and postoperative surface radiation. Often removal thyroid and cricoid cartilage. Also interstitial radiation.

LARYNX—Continued.

Clinic and Author	Number of Cases	Number Healed	Per Cent	Duration	Method
Stewart Harrison (Zurich)	11 (55% inoperable)	6	54%	Cases treated since 1929	Coutard.
Total all English Clinics Plus Quick's Statistics. All Carcinoma of Larynx.	351	99	28%	1 to more than 5 years	Surgery plus radiation—interstitial and surface.
Hegener (Hamburg)	23 { 4 suitable for thyrotomy; 7 suitable for hemilaryngectomy; 8 suitable for laryngectomy; 4 inoperable	9	39%	1 to 2 years	9—Radium per thyroid window. 4—Exclusive Coutard. 4—Coutard plus thyrotomy.

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LXIV

SOME OBSERVATIONS ON TEMPORAL BONE SECTIONS AND OTITIC MENINGITIS.*

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Literature abounds in excellent contributions relative to meningitis of otitic origin. Histologic reports proving the origin and pathway of the infection are comparatively rare. One finding, substantiating macroscopic observation made heretofore, was noted in a study of the horizontal serial sections of a right temporal bone in a case of pneumococcus type III meningitis.

The patient, white, female, age 7, entered with classic signs and symptoms of meningitis. The present illness began seventeen days previously with pain in and around the right ear, which subsided under treatment without incision of the drumhead. A year previously there was a discharge of moderate duration from the same ear. Examination showed the ear drums intact, not injected, and landmarks present. The right drum, however, was dull and slightly thickened. Impending respiratory failure contraindicated surgical intervention.

Fig. 1 (No. 4280), Sec. 230, represents photomicrographic proof of the otogenic origin of the meningitis. Examination of the cranial cavity failed to establish this fact. There is shown not only a direct anatomic connection between the mastoid cavity and the dura via the subarcuate fossa, but also a direct communication between the infected parantral cells and an epidural abscess.

Courville[1] refers to this appropriately as follows:

"A developmental process which is still in progress during infancy and childhood is the gradual obliteration of the fossa subarcuata. During this period an opening, situated above and

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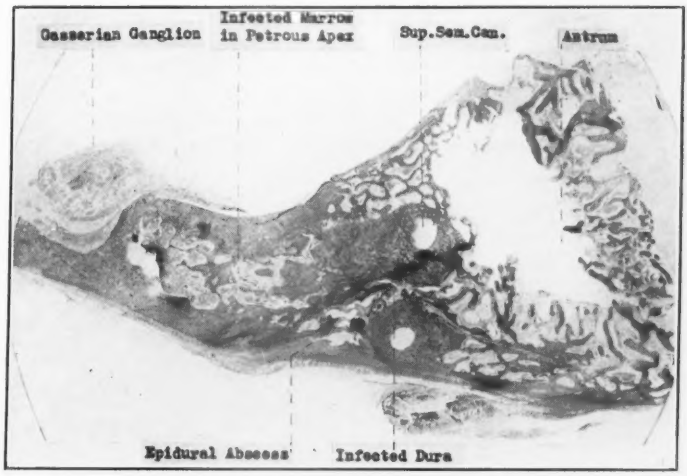

Fig. 1 (No. 4280), Sec. 230.—Right, white female, 7 years. Horizontal section at superior level of antrum showing epidural abscess at entrance of subarcuate fossa in direct communication with infected parantral cells.

behind the porus acusticus internus, represents a depression or even a canal which extends into the pars petrosa under the superior semicircular canal. Occasionally it may even approach the mastoid. A vein surrounded by a fold of pia mater sometimes extends into it. Purulent exudate has been traced from the mastoid to the cranial cavity through this canal."

Various textbooks on anatomy refer to a vein and an artery together with a process of dura, rather than "a fold or pia mater," extending into the fossa. D. Wolff, instructor in anatomy in otolaryngology, has noted in a previous study (observations yet unpublished) that in five cases the subarcuate fossa was found to communicate with the antrum, the dura actually serving as a lining of the antrum. This opportunity is taken to gratefully acknowledge the assistance of Miss D. Wolff in the detailed study of serial sections of temporal bones.

Higher magnification of the route of infection, as seen in Fig. 1, shows a definite infiltration of the dural tissue leading from the cranial cavity into the mastoid cavity, and with the epidural

abscess present, indicates that this was an extension of infection by continuity and not primarily infection of the meninges via the blood vessels.

Fig. 2 (No. 4098), Sec. 170, is taken from another series of sections in the files at the Oscar Johnson Institute, to show that the subarcuate fossa may remain patent at 48 years of age.

It is apparent that a patent subarcuate fossa would be conducive to the intracranial extension of infection in the event of a mastoiditis. Profant[2] minimizes the prominence given this route by Perkins,[3] and obviously this is but one of the various routes

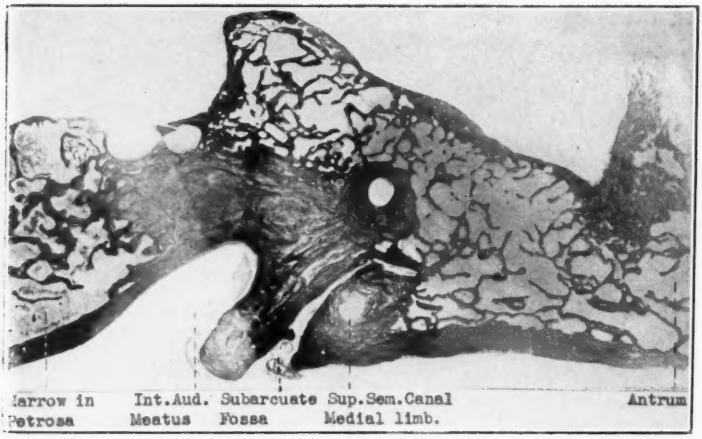

Fig. 2 (No. 4098), Sec. 170.—Right, colored female, 48 years. Horizontal section showing patent subarcuate fossa.

in which otogenous infection may involve the intracranial contents, whether it be by bone necrosis, through sutures, fractures, dehiscences, canals for nerves, perforating vessels or lymphatics.

Continuing with the original series, Fig. 3 (No. 4280), Sec. 531, shows the middle ear cavity proper remarkably free of disease but the parantral cells containing pus. The subepithelial tissue throughout is not greatly thickened, but it does show general round cell infiltration. The niche of the oval window is free of pus. Subsequent sections show infiltration present to a greater

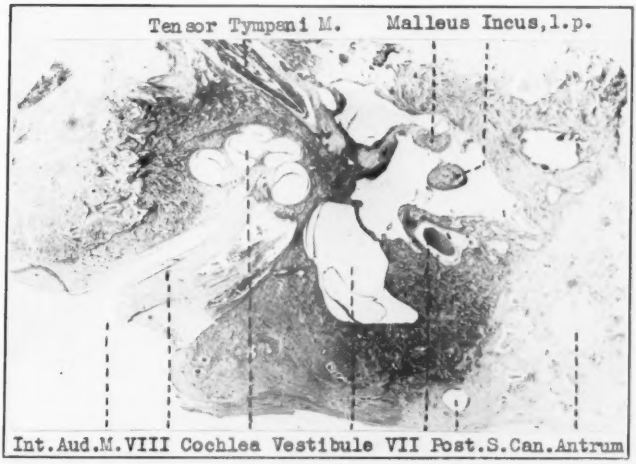

Fig. 3 (No. 4280), Sec. 531.—Showing infectious process practically healed in tympanum, but still active in parantral cells.

degree at the entrance of the eustachian tube, although it is patent as far as traceable. The inner ear shows evidence of an old serous labyrinthitis. There is a heavy infiltration along the eighth nerve.

Fig. 4 (No. 4280), Sec. 833, shows the fibrous layers and most of the cutaneous layer of the drumhead intact. The marrow in the petrous apex shows evidence of infection. Medial to the internal carotid artery and below the level of the round window is a mass of white diamond-shaped imprints, which have the appearance of cholesterin crystal imprints.

Wittmaack[4] and Crowe[5] picture similar cholesterin crystal imprints. Dean[6] states that "In sections of temporal bones with mastoiditis we often found diamond-shaped spaces in granulation tissue which contained cholesterol before it was dissolved out by the solutions used. There is no suggestion of cholesteatoma in or near these masses, so that in chronic suppurative otitis cholesterol in the ear is certainly not peculiar to cholesteatoma." Attention is again drawn to these imprints, although no conclusions are possible.

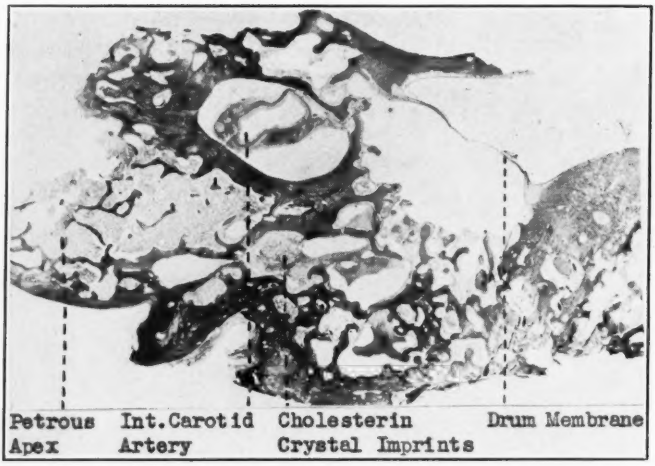

Fig. 4 (No. 4280), Sec. 833.—Showing lower level of middle ear cavity with fibrous layers and most of cutaneous layer of drumhead intact.

Photomicrographs from another series are next presented. The patient, colored, male, age 12, entered subsequent to the preceding case, with a definite meningitis following a bronchopneumonia, during which an acute otitis media on the right developed, the drainage stopping after one week of observation and treatment. Three weeks later neck rigidity was noted and lumbar puncture revealed increase of pressure, fluid turbid, 240 cells per cu. mm.; organisms could not be identified on the stained smear. On entry, the cell count was 5,000 per cu. mm., and organisms could not be located in stained smears. No history of previous ear involvement was obtainable. Otolaryngologic examination revealed the right ear drum slightly thickened; anteriorly and inferiorly there was a roughened area over which there rested a drop of colorless fluid. Perforation was not visible. A small hematoma noted on the anterior canal wall was considered secondary to previous manipulation. The only other deviation from normal was a slight pinkness in the posterior half of the drum. Spoken voice was heard in the right ear and caloric test showed nystagmus. The drum was incised, yielding only blood along incision. Culture

was taken. The left drum was intact, of normal color, with landmarks visible. The nose was anatomically good and not obstructed. The tonsils and adenoids were cleanly out and the posterior pharyngeal wall was not inflamed. Otologic surgery was deferred because of several considerations which must be mentioned. This patient entered the St. Louis Isolation Hospital subsequent to 457 other cases of meningitis, four-fifths of which proved to be meningococcus meningitis.

FOUR HUNDRED FIFTY-SEVEN CASES OF MENINGITIS: APRIL, 1929, TO APRIL, 1932.

Meningococcus	371
Tuberculous	32
Pneumococcus	26
Streptococcus	13
Type undetermined	9
Influenzal	3
Luetic	3

It has been impossible to demonstrate the meningococcus in stained slides of the cerebrospinal fluid in numerous cases of meningoccocus meningitis, but in pneumococcus and streptococcus meningitis the organism has practically always been shown.

Because of this inability to locate organisms in stained slides of this child's cerebrospinal fluid, and also in view of the apparent absence of exudate in the tympanic cavity, surgical intervention was deferred until report on the ear and spinal fluid cultures was possible. Antimeningococcic treatment was continued as meningococcus meningitis with a concurrent otitis was not beyond the realm of possibility. That a protective meningitis existed with no organisms demonstrable was the gravest possibility. Any treatment to be effective demanded immediate institution. The time element is again emphasized by Klemme[7] in reporting seven recoveries in forty cases of meningitis, other than meningococcus, when he states that "the average time that has elapsed between the recognition of the disease and institution of drainage in patients that have recovered was four and a half hours."

Examination early the next morning before reports were received showed the line of incision practically healed and no evidence of exudate in the tympanic cavity, and the entire drumhead

almost returned to normal except for slight thickness and pinkness in the posterior half. That the drum membrane picture is not always pathognomonic of the underlying pathology has been well shown histologically by Birsner and Wolff.[8] This fact is also manifest in the first case presented. Guggenheim and Ferris[9] in a report of four cases which they term dry necrosis of the mastoid have reviewed the possibilities, just as Hempstead[10] did in reporting six cases of definite mastoiditis in which the middle ear was

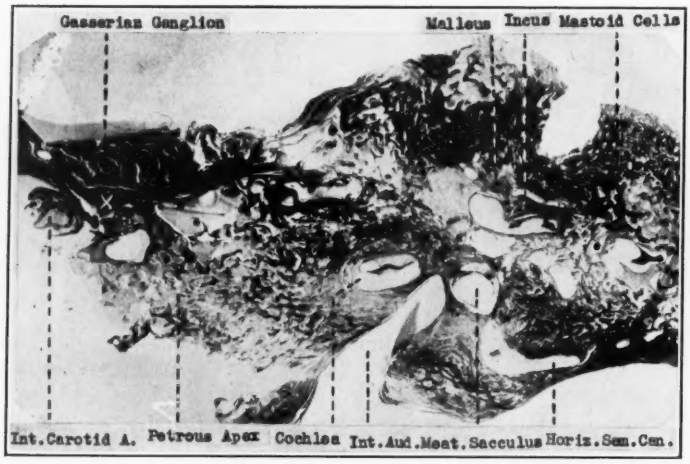

Fig. 5 (No. 24), Sec. 530-A.—Right, colored male, 12 years. Horizontal section at level of epitympanum. *X* indicates pus in carotid canal, with pathologic bony dehiscence, epidural abscess and entry of the infection through dura.

definitely not affected. There were, however, 500 cases in which the middle ear was obviously affected during the same period at the same clinic.

When the culture from the ear was reported negative, and the culture from the spinal fluid was reported positive for streptococcus, otologic surgery had been withdrawn from consideration. A neuro-surgeon performed a lumbar laminectomy but the child expired several hours thereafter.

Examination of the cranial cavity revealed no point of entry. The temporal bone on the right was removed for serial section, and the point of entry was located beneath the Gasserian ganglion.

Three pathologic dehiscences in the carotid canal are to be noted in the photomicrographs presented. Fig. 5 (No. 24), Sec. 530-A, shows a concentration of pus in the carotid canal beneath the Gasserian ganglion, higher magnification revealing pathologic bony dehiscence, epidural abscess and the point of entry of the infection through the dura.

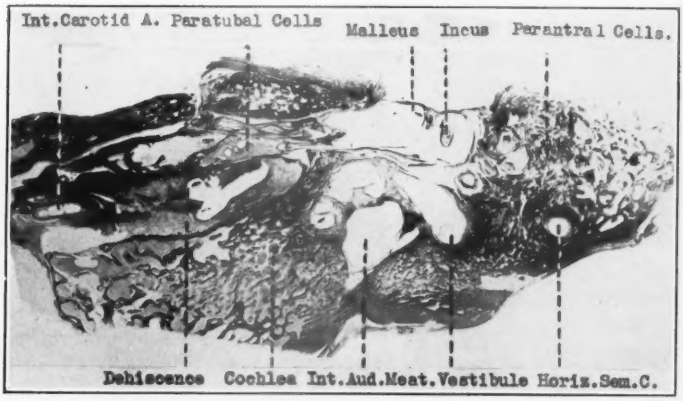

Fig. 6 (No. 24), Sec. 365-A.—Showing infection passing from paratubal cells into carotid canal through pathologic bony dehiscence. Note intact drum membrane and middle ear cavity practically clear of infection.

Fig. 6 (No. 24), Sec. 365-A, shows the dehiscence through which the infection entered the carotid canal from the paratubal cells.

Fig. 7 (No. 24), Sec. 151-A, shows the dehiscence which permits the infection to enter the apical cells.

Although the infection remains extensive throughout the mastoid and apical cells, it has cleared almost entirely from the tympanum, permitting the membrana tympani to remain intact and approximate the normal in appearance. The lumen of the eustachian tube is patent throughout, although the walls of the tube are infected. This patency apparently afforded sufficient drainage

of the tympanic cavity per se. Similar cases have recently been presented by Love,[11] in his macroscopic observations on the "complications of otitis media without rupture of the membrana tympani."

Little if any free pus is seen in the labyrinth except in the basal coil of the cochlea. The vestibular maculæ and ampullæ and the organ of Corti are in fair condition, but the basilar membrane and Reissner's membrane have failed to remain intact, indicating a probable early chemical change.

Fig. 7 (No. 24), Sec. 151-A.—Showing edematous granulation tissue, morphologically resembling mesenchyme, in direct continuity with abscess formation in petrous apex.

In addition to pus, granulation tissue is noted in the carotid canal and also in the apical cells. Somewhat removed from the point of abscess formation, as seen in Fig. 7 but in unbroken continuity with the granulation tissue walling off the pus, a tissue is noted which assumes the appearance of true mesenchyme. Dr. Howard A. McCordock, associate professor of pathology, in a personal communication, states that this tissue may "be regarded as similar to those seen in temporal bones of babies and young

children, as well as those reported a few times in adults." Crowe and Polvogt[5] have reported, but apparently in the presence of infection, that embryonal tissue may occasionally be present in the middle ear and mastoid past the age of 50. Marx[12] feels that this tissue may be secondary to infection occurring in adult life with a reversion of adult mucosa to its primitive form, but according to Kopetzky,[13] pathologically such a reversion of well organized tissue to one of embryonal type, owing to suppuration late in life, has never been proved.

Dr. McCordock's description of the tissue in question follows:

"In several sections from the lower portion of the temporal bone there is a large cavity in the petrous apex, irregular in size, and filled with a loose myxomatous type of connective tissue resembling the mesenchyme found in the embryo. This primitive connective tissue consists of irregular, fusiform cells that have long, slender processes connected with one another. The spaces that these connecting processes enclose are in many places filled with a homogeneous intercellular substance, staining faintly with eosin. However, many of these intercellular spaces are clear, and if they contained liquid or semiliquid material in the fresh state it has been dissolved by some of the reagents used in preparing the sections. A moderate number of large and small mononuclear wandering cells are scattered throughout the tissue. One end of the mass of primitive connective tissue is continuous with an area of acute inflammation in a neighboring tissue, and in this region it is densely infiltrated with polynuclear leucocytes. Thin walled blood vessels extend throughout this loose connective tissue, and those near the acute infection are surrounded by masses of polynuclear cells. Elsewhere the vessels show no signs of inflammation and are not so numerous as they usually are in granulation tissue.

Special stains for fibrous tissue reveal a delicate staining of the connective tissue fibrils: red with van Gieson's stain and blue with Mallory's stain. In this respect the fibrils stain the same as those of ordinary connective tissue. There is, unfortunately, no staining method for differentiating the embryonic type of connective tissue known as mesenchyme from the ordinary fibrous tissue formed as the result of many types of injury to adult tissues."

In certain areas this loose myxomatous type of connective tissue contains directly in it a mass of pus. This fact, together with its unbroken continuity with definite granulation tissue and pus, would indicate that this mesenchyme-like tissue is nothing more than granulation tissue which has become edematous.

CONCLUSIONS.

1. Serial sections of temporal bones are necessary at times to prove the otogenic origin of meningitis.

2. The otogenic meningitis may exist, although the drum membrane, as a result of drainage via a patent eustachian tube, may approximate the normal in appearance.

3. The subarcuate fossa may be patent at the age of 48, and when patent it is conducive to intracranial extension of infection from the mastoid.

4. An edematous type of granulation tissue, morphologically resembling mesenchyme, may be present at the age of 12 in an infected temporal bone.

BEAUMONT BUILDING.

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LXV

ADVANTAGES OF ONE-STAGE LARYNGECTOMY THROUGH A STRAIGHT MIDLINE INCISION IN CASES OF INTRINSIC CANCER.

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Carcinoma of the larynx is one of the most hopeful types of cancer with which the surgeon has to deal today, and constant improvement in the technic of laryngectomy is making the prognosis increasingly favorable. In the days of Billroth and Langenbeck the mortality was staggering, and the percentage of recurrences discouragingly high. Today the mortality is not more than 5 per cent in the hands of skilled surgeons. In 1929 Mackenty reported a series of 230 cases with a surgical mortality of less than 3 per cent. Recurrences in the intrinsic cases, to which I shall confine this discussion, are rare.

For several years it was my privilege to be associated with the late Dr. Mackenty, whose work marked such an outstanding advance in the field of laryngectomy. While watching the postoperative progress of patients operated on through his T-shaped incision, two unfavorable points in an otherwise excellent technic impressed me more and more. Although his mortality was low and the late end-results good, one could not fail to note the disadvantages, even in the hands of a master surgeon, of the angular and poorly nourished flaps caused by this incision, particularly as the method of drainage put still further obstacles in the way of primary union. Retraction of the cut muscle and the malnutrition of these right-angled flaps were a frequent cause of delay in union, with an augmented chance of infection.

Keeping open for drainage the lower portion of the wound, a region which has been aptly called the "danger zone" in these cases, was a second feature of the Mackenty technic which seemed particularly undesirable. It should be remembered that any wound contaminated by saliva is prone to slough, and to keep a wound

having a large communication with the pharynx free from infection presents a real problem to the surgeon. If he gets sloughing between the hypopharynx and the trachea, he is confronted with a delay of many days and possibly a plastic.

Prompt closure by primary union of the skin, including a bridge of deep tissues between the trachea and the upper drainage tubes, would render such catastrophies as hypopharyngeal fistula much less likely, and would also block off droppings from the pharynx and hypopharynx which so readily infect the trachea and lungs. It also would make possible removal of the feeding tube at a much earlier date.

After due consideration of the problems involved, I have, therefore, worked out a modification of the Mackenty technic, abandoning the T-incision for a straight midline incision, and using short lateral incisions, parallel to the midline, for drainage. These do not interfere with primary union of the skin in the whole length of the wound and of the deep tissues between the trachea and the upper drains, which is so desirable. The drainage is equally adequate, and the convalescence of patients operated on by this method has been remarkably shortened—the feeding tube being removed, as a rule, between the eleventh and fourteenth day.

The technic which I now use differs from that of Dr. Mackenty in two essential points: the manner of incision, and the method of drainage.

TECHNIC.

Intratracheal inhalation anesthesia is used.

The oropharynx is packed off with gauze, which takes care of a lot of saliva and secretion that would otherwise contaminate the operative field.

Using a general anesthetic from the beginning saves anesthetizing the patient with the wound open, as was done with the Mackenty technic. By the latter method, when you change from local to general anesthesia, the wound is wide open, and although the trachea is plugged with the conical rubber tube there is usually quite a little straining during the induction of the anesthetic and, with this, often more or less bleeding from the wound. This in-

creases the danger of getting blood into the lungs and also augments the possibility of the wound becoming infected.

When the patient is under the general anesthetic from the beginning there is no delay for the change of anesthetic, and the dangers noted above are to a large extent obviated. The advantages to both patient and surgeon are apparent. I believe that this method, except where there are definite contra-indications, is much preferable to local, or to a combination of local and general. It shortens the time of operation, avoids the infiltration of tissue which diminishes resistance to infection, and does not subject the patient to drugs and narcotics approaching the physiologic limit as is often necessary when local anesthesia alone is used (morphin, scopolamin, adrenalin and novocain). As a matter of fact, very few patients can tolerate the operation under local alone, and one is faced with the necessity of adding ether to an already heavy load of drugs.

The larynx is exposed through a straight midline incision beginning just below the hyoid bone and extending to the second ring of the trachea. This straight midline incision gives perfectly adequate exposure for skeletonization of the larynx in all intrinsic cases. Any slight advantage which the T-shaped incision may have in the way of exposure is more than offset by disadvantages when it comes to postoperative healing and the dangers of infection, points which will be discussed in detail later.

When the larynx has been skeletonized, the anesthetist removes the gauze from the oropharynx and all secretion is sucked out. Then any secretion that might be in the trachea is removed by suction through a rubber catheter, and the intratracheal inhalation tube is also removed, after which the trachea is cut through to the anterior esophageal wall and partially separated posteriorly. Then the Mackenty conical rubber tube is placed in the tracheal opening and the anesthetic is continued through this. A strip of iodoform gauze is packed through the larynx into the hypopharynx.

From the time the trachea is cut, it must be watched carefully in order to prevent any blood from being aspirated to the lungs

during the operation. In case any blood should get into the trachea, it should be sucked out with the rubber catheter immediately.

I now proceed to remove the larynx in a manner very similar to that described by Mackenty:

"The larynx is separated from the esophagus from below upward to a point behind the arytenoids, the degree of detachment depending on the extent of the neoplasm. It is then allowed to fall back into position and the thyrohyoid membrane is divided, thus opening into the hypopharynx below or above the attachment of the epiglottis as indicated by the extent of the disease. If the growth is found to be entirely intrinsic, the larynx is removed by cutting as close as possible to the superior border of the thyroid cartilage. The opening thus made in the hypopharynx is small and lends itself better to successful repair. If the disease has approached the top of the laryngeal box or has involved the arytenoid, then more tissue is sacrificed."*

As soon as the larynx is removed, the esophageal opening and that in the hypopharynx are closed, except for a small aperture to aid the surgeon in guiding the feeding tube into the esophagus.

A feeding tube which has been tested for patency is then introduced through the nose and passed downward. When it comes in view through the small opening in the esophagus, eight inches is measured off on the external portion of the tube from the tip of the nose, and the surgeon continues to guide the tube into the esophagus until this eight-inch marker has been reached. The tube is fixed in this position and further egress into the esophagus prevented by wrapping sufficient adhesive tape around it at the marker to make passage into the nose impossible. By means of a silk string the external portion of the tube is fixed to the forehead.

Closure and Drainage.—As soon as the feeding tube is in place we complete the closure of the esophageal opening and hypopharynx with No. 1 catgut. Then the tracheal tube is removed, and the trachea is attached to the skin by means of interrupted Equisetene sutures.

*From Mackenty, J. E.: "Cancer of the Larynx," p. 41.

Before closing the midline two small lateral incisions for drainage, about three-fourths of an inch long, are made parallel to the upper portion of the wound and just anterior to the sterno-cleido-mastoid muscle. (See Fig. 1.)

Two other lateral incisions large enough to admit a double drain* are made opposite the trachea, just anterior to the sterno-cleido-mastoid, for drainage around the lower part of the wound. A small rubber catheter drain tube with three openings in the

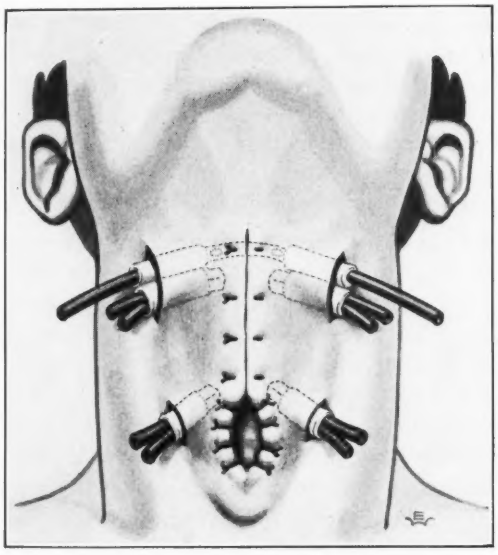

Fig. 1. Method of drainage.

center, close enough together to drain the center of the wound, is placed through and through the upper incisions from one side to the other of the deep wound (tube z. x. 16 Fr., 8 Eng., 11 Am.). A double Mackenty drain tube wrapped in gauze and open only at its distal and proximal end is placed in each upper lateral incision, just below the through and through tube, and is pushed in near to the center of the wound.

*In some instances a single drain will suffice.

In each of the lower drainage incisions a double rubber drain tube wrapped in iodoform gauze and with an opening about 1 cm. from the distal end, is placed to the bottom of the wound, pointing slightly inward and upward (Fig. 1).

The midline incision is then closed with parallel mattress stitches of heavy silkworm gut, following the Mackenty technic. These are usually placed between 1½ and 2 cm. apart. The skin is then closed with dermal sutures (Fig. 1).

The trachea is taken care of in the same manner as Mackenty describes. A large tracheal cannula (36) is wound round with gauze impregnated with bismuth paste. The winding is done in such a way as to form a conical cork, which is inserted into the trachea and should fit as a cork does a bottle. The object of corking the trachea is to prevent tracheal secretions from contaminating the wound, and wound secretions from entering the trachea. The cannula is left in place, usually from four to five days. However, in case of coughing or the accumulation of secretions, it may be necessary to remove it and investigate the cause before that time. After the first five or six days, paraffin gauze is substituted for the bismuth paste. This cannula is continued until the wound is entirely healed.

The wound is dressed with wet compresses (bichlorid solution 1/5000).

Postoperative Care.—This is a very important stage of laryngectomy because the anatomic position of the wound lends itself to all sorts of complications in the way of infection. It is difficult, however, to lay down general rules, inasmuch as the care varies widely with the individual case.

The drainage tubes must be watched carefully to see that they are open and should be tested at each dressing, and oftener in case there is any secretion from the wound. In the latter instance, the tubes should be allowed to extend through the dressing in order that the nurse shall be able to keep them clean by dry suction.

The frequency with which dressings are changed depends upon the condition of the wound and the temperature—as a rule, once a day for the first three days unless some special reason makes more frequent inspection advisable. Beginning with the fourth

day postoperatively, the dressings should be done twice a day if for no other reason than to inspect the tissues adjacent to the wound, in order to keep ahead of any possible infection which may arise. The fourth to seventh days may be regarded as the critical period in postoperative care, although infection in these cases is very insidious and may appear at a later date. One must, therefore, always be on guard.

If everything comes along well, and there is no infection or sloughing, the side drains next the through and through tube are slipped out, usually about the seventh day. The lower drainage tubes are removed gradually, according to the condition of the lower part of the wound, especially around the trachea—usually about the eighth or ninth day. But one cannot lay down a definite rule. He must be governed by the condition of the wound, and this is where experience is invaluable. The through and through tube is left in until there is practically no secretion or exudate draining from the wound. After removal of the through and through tube, as soon as the patient is able to swallow fluid without a leakage, the feeding tube is removed.

The upper lateral incisions usually close within forty-eight hours without suture. One has to be very careful not to remove these tubes too soon, on account of the anatomic position—the openings in which they are placed are slit-like, between the muscles, and collapse and close very quickly.

Postoperative Voice.—This is a question which concerns every patient with a laryngectomy. By continued effort and careful training it is possible for the intelligent patient to acquire a very satisfactory voice. I have a patient, operated on four years ago, who returned to his work as a salesman on the road, five weeks after operation, with a very satisfactory voice, and is still doing the same work successfully.

DISCUSSION.

From the point of view of both patient and surgeon, I believe the method above presented has distinct advantages:

1. Much less time is consumed in securing hemostasis when using the straight incision and staying close to the larynx and trachea. By omitting the horizontal section of the T, very few

vessels are cut and practically no large ones. I have used as few as six or eight ligatures. By minimizing the number of vessels cut, a great reduction in the danger of postoperative hemorrhage is gained, and the postoperative nutrition of the tissues around the incision is much greater than when the incision extends across the neck with a large amount of consequent bleeding. Obviously the better the nutrition, the earlier the union.

Mackenty himself, both in his writings and in the clinic, tacitly admitted the difficulties of obtaining prompt union at the angles of the T-incision. I will quote one paragraph: "It is essential to get a primary union at one point—that is, where the two lines of the T cross. I have observed that if the integrity of this part of the wound can be maintained, the subsequent healing is much more rapid and a hypopharyngeal fistula does not form. If a break occurs at this point, or if the wound has to be entirely opened to secure better drainage, an effort should be made, as early as seems prudent, to bring back the angles of the T into place. . . ."

With the straight incision, the surgeon's worries implied in these "ifs" are to a large extent removed by primary healing and by the elimination of poorly nourished flaps.

2. A second advantage of the straight incision is minimum interference with the muscles of the neck. There is no horizontal section of muscles except those attached to the larynx, thus eliminating the immediate and untoward tendency of cut muscle to retract, often leaving not much but skin near the suture line. In making the small lateral incisions for drainage, no muscles are cut, the knife being directed through the fascia and between the muscles.

3. Early firm union with a healthy bridge of deep tissue in the danger zone between trachea and hypopharynx is a great advantage, in that it prevents the secretions from the hypopharynx from dropping into the trachea—a constant source of danger and trouble when a fistula extends from the hypopharynx to the trachea.

4. The drainage being dependent and parallel with the bottom of the wound, the accumulation and retention of pent up secretion is much less likely.

5. The earlier removal of the feeding tube made possible by this method is greatly appreciated by the patient and adds materially to his comfort. I recently discharged a patient whose tube was removed on the eleventh day following operation. He was able to swallow liquid on the ninth day without a leakage, and left the hospital two weeks after operation.

6. My method of closure eliminates a long period of irrigation and thereby materially shortens the period of hospitalization and nursing care.

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LXVI

DIOTHANE AS A LOCAL ANESTHETIC FOR THE NOSE AND THROAT.

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The excellent results obtained with diothane as a local anesthetic for the mucous membrane of the urethra,[1] [2] have naturally suggested its use in the nose and throat.

This report is based upon a large number of cases in which diothane has been used by the writer during the last year.

After preliminary work to discover the proper concentration of the anesthetic to use in the various cases and to perfect a somewhat new technic for its use, it was found that for topical application cocain could be entirely replaced by diothane and that the resulting anesthesias were at least equally satisfactory. Statistically there were no more, or perhaps fewer failures with diothane than with cocain, and occasional failure could be directly attributed to variations from the necessary technic. The most marked difference in technic between the use of diothane and the use of cocain are the necessity for a slightly more prolonged application of diothane, and the possibility of working under diothane anesthesia for longer periods or at longer intervals after the anesthetic is applied.

Irrigation of the nasal passages with Bledsoe Fisher solution[3] has required a distinct anesthesia to prevent a rather intense pain due of course to the hypertonicity of the solution. Diothane in a concentration of ½ to 1 per cent has been applied to one and one-half inch cotton pledgets which were inserted deeply into the middle meatus for from five to ten minutes. The shrinking of the mucous membrane and the resultant anesthesia are as satisfactory as was previously obtained with 1 to 2 per cent cocain. The onset of anesthesia was slightly slower than with cocain, although the duration of anesthesia appeared to be definitely longer. These differences led to adopting a technic whereby the anesthetic was

applied and the patient allowed to rest with the cotton pledget in place for at least ten minutes. There was little danger of finding the anesthesia waning if the period were more extended, as is so frequently the case when cocain is used. Several hundred cases in which diothane has been used have convinced the writer that diothane has given better results for this purpose than cocain.

The topical application of diothane solution by means of a spray to the pharynx and larynx introduces a new problem. Since the anesthesia develops somewhat slowly the spray application is less satisfactory, due to the relatively short time during which the solution remains in contact with the mucous membrane surface. In order to offset this shorter contact it was found advisable to increase the strength of solution. The solubility of diothane in distilled water is limited to approximately 1 per cent, but supersaturated solutions can be prepared which do not crystallize for several hours. For spray purposes a 2 per cent solution has been used. This solution as furnished contained precipitated crystals. By means of gentle heat the crystals were redissolved and the resulting solution was found to be stable, even after cooling, for several hours. Neither this solution nor more dilute solutions should be allowed to remain in contact with the metal tube of the spray, and it is not advisable to keep the solution in flint glass bottles since either the metal tube or the alkaline glass will in-time act on the solution causing a precipitation and loss of anesthetic power. The solutions are, however, stable indefinitely if stored in nonalkaline glass containers.

While the use of a 2 per cent solution has given somewhat more rapid anesthesia, the 1 per cent solution has proven to be satisfactory when carefully applied and is much easier to use. In general the 1 per cent solution should be frequently applied and held in contact with the tissues for as long a period as is feasible.

Satisfactory anesthesia for bronchial lavage (fifty cases) and bronchoscopy (five cases) has been obtained with a 1 per cent solution of diothane. This solution was sprayed over the throat two or three times at five minute intervals, a total of 4 to 6 cc. being used for each case. The resulting anesthesia was at least equal to that obtained with 5 to 10 per cent cocain.

The application of the 1 per cent solution of diothane on a cotton pledget beneath the inferior turbinate for the purpose of sharp needle puncture of the antrum produces the requisite anesthesia about as rapidly as cocain. The 2 per cent solution used in this manner produced a satisfactory anesthesia with less delay. Sufficient anesthesia for antrum lavage through the natural or accessory osteum has been obtained by applying 1 per cent diothane on a cotton pledget in the middle meatus for ten minutes. It seems to be advisable to rub some of the solution into the membrane with a probe after removing the pledget.

Diothane has served admirably to relieve certain types of neuralgic pain by anesthetization of the sphenopalatine ganglion. A cotton-tipped probe saturated with the 1 per cent solution and placed over the ganglion was allowed to remain in place for fifteen minutes. This treatment produced a prompt relief from pain and appeared to have a definitely longer effect than 10 per cent cocain similarly used.

Since diothane had proven to be unusually potent it was decided to try it for anesthetizing the ear drum prior to myringotomy. Preliminary experiments in rabbits showed that when a cotton pledget soaked in 2 per cent diothane was placed against the ear drum for fifteen minutes, a sufficient anesthesia resulted to allow painless paracentesis. After a series of experiments showing consistent results and checking the fact that a similar anesthesia could not be obtained with much stronger cocain solutions, the same technic was tried in humans, and while complete freedom from pain was not achieved it was found possible to obtain enough anesthesia to permit paracentesis provided that the technic was sufficiently delicate. A series of several cases have convinced the writer that diothane anesthesia is sufficient for a skillful myringotomy and that the use of phenol or an inhalation anesthetic may be avoided. Diothane makes the ear drum gray and lusterless during the anesthetic period and appears to be without effect on healing after puncture. A 5 per cent solution of diothane in benzyl alcohol has recently been used and has proven to be more satisfactory than the aqueous solution for this purpose.

The development of anesthesia after application of diothane to mucous surfaces is accompanied by a relatively slight degree

of blanching of the tissue. No toxic or other undesirable effects have been noticed, and the headaches and giddiness which so often follow the use of cocain were entirely lacking. Several patients who were known to be sensitive to cocain have been treated under diothane anesthesia and careful observation has shown a total lack of reaction. The patients themselves remarked that they did not feel the usual reaction and asked whether or not cocain had been used.

In patients who do not show any reaction to cocain there is no noticeable difference in results when diothane is used. In a series of cases where the antrum has had to be irrigated from time to time diothane, 1 per cent, has been used alternately with 10 per cent cocain without the patient knowing that a change had been made.

Since diothane has proven to be free from toxicity in a comparatively large series of cases, the 1 per cent solution has been furnished to several patients following tonsillectomies. These patients sprayed small quantities over the fauces from atomizers. Complete relief from pain resulted for periods of from one and one-half to three hours, and the spray was repeated at corresponding intervals. Frequent observations of these patients indicated that this treatment with diothane had produced no deleterious effect and had not delayed healing. The writer believes that this method of relieving postoperative pain is much less harmful than the current practice of gargling a suspension of acetylsalicylic acid. In this connection it is to be noticed that diothane has a quinin-like taste, although it is not a quinin derivative.

SUMMARY.

As a result of this study, the conclusion has been reached that diothane, properly used, is a satisfactory local anesthetic to replace cocain for all routine uses in the nose and throat. Diothane may be used in considerably lower concentrations than cocain, although the onset of anesthesia is somewhat slower. The resulting anesthesia is considerably more lasting than that produced by cocain and frequently much more striking. Its action on the ear drum allows an almost painless paracentesis. In a series of several hundred cases in which diothane has been used there have been no

signs of toxicity and diothane has not produced the undesirable reactions sometimes found with cocain. Diothane is safe for routine use as a spray to relieve posttonsillectomy pain and is preferred for this purpose to acetylsalicylic acid.

656 Doctors' Bldg.

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LXVII

PANTOCAIN AS A SUBSTITUTE FOR COCAIN: PRELIMINARY REPORT.

ALEXANDER F. LASZLO, M. D.,

NEW YORK.

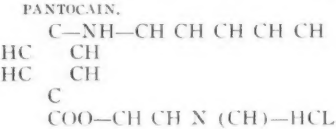
Cocain is still one of the most important drugs used in nose and throat practice and up to the present time has had no substitute. Many rhinologists are still using 10 per cent cocain even for diagnostic purposes, which can be replaced by a 2 per cent solution plus phenol (Geshe)[1]. Butin, tutocain, nupercain and phedrocain, which for the past few years have come under my observation, do not compare with the properties of cocain, and although there are some who still use them, they have never attained the popularity of cocain because of their many disadvantages.

About a year ago my attention was first called to a new drug which was claimed to have merits above all the drugs previously mentioned, plus all the advantages of cocain without its habit forming quality and unstability. In addition, it is materially cheaper. This drug is called "pantocain." Experimental work was done in the Nose and Throat Clinic of the New York Postgraduate Hospital of the Columbia University Medical College. My experience with the drug is purely clinical, and as to the laboratory reports I rely entirely on the experiments of the German authors, Fusganger-Shauman[2] and H. Schmidt.[3]

Pantocain is a novocain derivate:

NOVOCAIN.

PANTOCAIN.



It is a white crystalline powder, easily soluble in water, saline or alcohol; has a slightly bitter taste. The specific gravity in effective solution is less than that of the spinal fluid.

The law which was set by Braun for a good local anesthetic, especially as a substitute for cocain, is still in effect and in judging any drug we have to adhere to his principles.

1. The drug used as a substitute must be less toxic and have more or the same anesthetic effect. Inasmuch as pantocain can be used both for injection or topical application, the comparison must be made from these two viewpoints.

As we see from Wiedhopf's[4] extensive experiments, comparing the action of novocain, pantocain, nupercain and tutocain, he found that the pantocain in one-tenth of 1 per cent solution is a much more effective anesthetic than either novocain or tutocain of the same strength. The duration of the anesthetic effect was longest with nupercain; then follows pantocain, tutocain, and last novocain. The concentration of the drugs tested was the same as that usually used in practice: one-tenth of 1 per cent nupercain, 1 per cent novocain, and 1 or one-half per cent tutocain. As regards their toxicity, he experimented with dogs, gave the drugs intravenously, watching the blood pressure, breathing, and the heart condition, and found that with an injection of one-tenth of 1 per cent pantocain, 4 mg. per kg., the blood pressure fell from 110 to 70, the pulse was very little changed, and after a few minutes (three and one-half) was perfectly normal. With nupercain the same experiment showed a drop in the blood pressure from 70 to 20, which was returned to normal very rapidly; but a damage to the heart action became very noticeable with the symptoms of irregular and deep breathing, cramps and narrowed pupils. This condition prevailed for about twenty minutes. With tutocain, where 1 per cent solution is usually used, 40 mg. per kg. was injected, which resulted in a drop of the blood pressure from 80 to 20 at the beginning, without any damaging symptoms to the heart action. However, a weakening of the heart developed suddenly later on. Breathing stopped so that artificial respiration had to be resorted to. The blood pressure partly returned, but never to the previous normal. Although Weidhopf claims that

the same experiment cannot be applied to a human being unconditionally, he still contends, with Smith and Runge[5] concurring, that pantocain causes the least changes in the heart action and at the breathing center.

Experiments with guinea pigs show the lethal dose to be approximately:

	Novocain	Cocain	Nupercain	Pantocain
Intravenous	55-60[A] mg. per kg.	7.5[BD] mg. per kg.	2— 4[D]	6-10[E]
Subcutaneous.....	450[B] mg. per kg.	75[C] mg. per kg.	5—10[D]	20-30[E]

The same findings were reported by Lundy and Essex[6] from the Mayo Clinic.

From all these experiments we may conclude that the absolute toxicity of pantocain and cocain is about the same; pantocain even somewhat more. However, inasmuch as pantocain is at least ten times as powerful as cocain, the relative toxicity will be much less. The same conclusion also shows that the relative toxicity of novocain is much less than that of pantocain.

H. Schmidt found by comparative testing that pantocain is most effective when dissolved in distilled water, novocain in Braun's solution and nupercain in normal saline.

I am using one-tenth of 1 per cent pantocain in distilled water for tonsillectomies and for submucous injections, with one drop of adrenalin to a dram; waiting three or four minutes, we get an absolutely painless operative field, with up to now no complications or reactions such as fainting spells and pallor, which are very often observed with the injection of novocain. However, the advantage of the somewhat longer lasting anesthetic effect as compared with the relative toxicity of the drug would not warrant the replacing of a perfectly harmless and inexpensive drug such as novocain, which has proved its worth.

2. The drug should be readily absorbed by the mucous membrane.

Runge tested the comparative strength of cocain, nupercain and pantocain on the cornea of a guinea pig and found that 1/4000 cocain does not give any anesthesia; nupercain gives an anesthesia which lasts for sixty minutes, and pantocain for thirty or forty

minutes. In 1/1000 solution the cocain gives an anesthesia for ten minutes, nupercain for 130 minutes and pantocain for sixty minutes. He also tested the anesthetics on the frog's foot and found that the anesthetic strength of 1 per cent pantocain is about the same as 2 per cent nupercain or 10 per cent cocain, and that the anesthesia lasts longest with nupercain and shortest with cocain.

	Nupercain	Pantocain	Cocain
1:250	After 6 minutes no anes.	After 3 minutes beginning of anes.	No anes.
	After 9 minutes beginning of anesthesia	After 6 minutes definite anes.	No anes.
	After 20 minutes definite anesthesia	After 9 minutes complete anes., which after 15 minutes slowly decreased	No anes.
	After 24 minutes complete anesthesia		
1:150	After 6 minutes no anes.	After 3 minutes strong anes.	After 30 minutes no anes.
	After 9 minutes strong anes.	After 6 minutes complete anes., which after 20 minutes slowly decreased	
	After 12 minutes complete anes., which after 20 min. slowly decreased		
1:100			After 20 minutes anes.

Runge tested the same drugs in the posterior pharyngeal wall and found that the 2 per cent pantocain gives a much better anesthesia than the 10 per cent cocain or 2 per cent nupercain, and in these concentrations the anesthesia begins quickest with the cocain, is almost the same with pantocain, and starts the latest with nupercain.

3. The drug should not cause any damaging effect to the tissues to which it is applied. No reaction of any kind could be noted by me with the use of pantocain, and, as the colored photographs of Wiedhopf's experiment show, the subcutaneous injection of pantocain one hour after the injection gave the same reaction as the novocain as compared with nupercain and tutocain which gave more reactions.

One of the most desirable qualities that cocain possesses is that upon application to the mucous membrane it causes ischemia.

Although pantocain does not produce any constriction of the small vessels, no hyperemia can be observed either, and Runge is inclined to think that some anemia can be observed if a 2 per cent solution of pantocain is used. I could not see any anemia while I was using the drug in this percentage, but combined with adrenalin it produced the same ischemic effect which is caused by cocain with the same amount of adrenalin. Therefore, more profuse bleeding could not be noticed (submucous resections). Neither could any other postoperative reaction be seen (unusual swelling, hematomas or hemorrhagic infiltrations).

4. The drug should be soluble in water and constant in solution for sterilizing purposes. This rule is easily applicable to pantocain, inasmuch as it is very soluble in water, in normal saline or in alcohol, and will withstand boiling for a long period without any decomposition. It is unstable in alkalin solutions but not as much as nupercain. It is much more stable than cocain or novocain and in the cold will remain so for an indefinite period of time. The syringe which is used must be free from soda solution. Therefore it is best to sterilize only with alcohol.

5. It should be miscible with adrenalin and ephedrin. Pantocain as well as cocain and nupercain can be easily mixed with adrenalin and ephedrin.

As the foregoing shows, we are dealing with a drug which fulfills all the requirements of Braun's rules, and therefore enables us to replace the cocain entirely. However, I am not at all in favor of replacing novocain, because the relative toxicity of pantocain is still higher than novocain, and there is only a small advantage to be gained, with a somewhat longer lasting effect of the anesthesia. As a substitute for cocain, in my opinion, we are dealing with a drug which in the useful strength is five or ten times less toxic, not habit forming, more stable and economical.

For the past few months we have substituted the 2 per cent pantocain for topical cocain application with excellent results. We are saving a great deal of time, because after the packing of the nose we do not have to wait longer than three or four minutes, compared with the fifteen or twenty minutes which are required by the Geshe solution. Polypectomies, turbinectomies and

antrum lavages were done with perfect results by only swabbing the operative area a few times. The pharynx and larynx react faster than to 10 per cent cocain. I had occasion in one case to use 2 per cent pantocain in the larynx at one time and 10 per cent cocain with a preliminary injection of one-fourth gr. morphin at another. The patient expressed the opinion that the first manipulation was just as painless as the second. A surprisingly good result was obtained in cases of peritonsillar abscesses, where we know the absorption of a drug is very poor, making the operation usually a very painful procedure.

I have used pantocain in the 2 per cent solution with excellent results in electrocoagulation of tonsils and electrocoagulation of turbinates. There was a definite advantage noted when we used it in the nose without adrenalin for removal of polyps and resection of the posterior tip of the inferior turbinates where no shrinking of the tissues should follow the application of the anesthetic.

Dr. Ernst,[8] of the Surgical Polyclinic of Munich, calls attention to a very peculiar finding. He used pantocain (one-tenth of 1 per cent) in twenty-four goiter operations, and although his technic was exactly similar to the one he used with novocain, he noticed complete loss of the voice immediately following the injection, before the operation, in seventeen cases. Normal voice returned, however, after one or one and one-half hours. Inasmuch as he had a similar occurrence with novocain in only about one-half of 1 per cent of his cases, he is inclined to believe that the nerve substance possibly has a higher sensitivity to the drug, which causes this reaction, or that some special biochemical condition is present which makes it possible for the anesthetic to reach a deeper surface. He observed in this same operation that a much longer anesthetic effect is produced with the use of pantocain.

CONCLUSION.

The foregoing leads us to conclude that pantocain is less toxic, more stable than cocain, and possesses many other advantages over it.

Novocain, for infiltration, rates far above pantocain, as it is less toxic and more economical than the latter.

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NOTICES.

THE AMERICAN BOARD OF OTOLARYNGOLOGY.

An examination was held in Milwaukee, Wis., June 12, 1933, during the meeting of the American Medical Association. Forty-eight candidates were examined, of which nine were conditioned or failed.

The Board will hold an examination in Boston, September 16, 1933, just prior to the meeting of the American Academy of Ophthalmology and Otolaryngology. Prospective applicants for certificates should address the Secretary, Dr. W. P. Wherry, 1500 Medical Arts Building, Omaha, Neb., for proper application blanks.

Clinical Notes and New Instruments.

LXVIII

CARCINOMA OF NASOPHARYNX WITH DESTRUCTION OF THE CRANIAL NERVES, INCLUDING THE SECOND AND EIGHTH NERVES.*

HOWARD C. BALLENGER, M. D.,

CHICAGO.

T. R., a male, aged 40, first consulted me about glands of his neck in August, 1928. He gave a history of having had a gradual growth of the anterior cervical chain of glands of the left side of the neck beginning at least a year before (August, 1927) the time he consulted me. About six months before he had had the glands removed from the left side of the neck. In the meantime the glands on the right side had grown rather rapidly, but without glandular pain or temperature.

These glands at the time I examined them were matted, hard and immovable. Each gland was about the size of an English walnut.

In searching for a possible original focus in the nose or throat, a very small papillomatous swelling was found in the left fossa of Rosenmüller, very near the orifice of the eustachian tube. There was a slight infiltration of the surrounding tissue but not enough to impinge upon the lumen of the eustachian tube, as he had no tinnitus, fullness or pain in the ear at this time.

The glands in the neck were removed by dissection and without recurrence until a short time before his death, a period of about four years.

According to Broder's system, the carcinoma at that time was classed as grade IV, which would mean, if we accept Broder's system of grading, that the growth would be most susceptible to radium and X-rays and the least susceptible to surgery. Consequently radium and X-rays were given as the treatment of choice.

The patient chose to go to the Mayo Clinic for these radium and X-ray treatments, which were given every six to twelve weeks (except the past six months) by Dr. G. B. New.

The gradual extension of the growth into the base of the brain also produced a gradual development of his symptoms, depending upon which of the cranial nerves were involved.

The earliest and most pronounced and most persistent symptom was the neuralgic pain in the face due to involvement of the fifth nerve and the Gasserian ganglion. From a clinical standpoint the sixth and second nerves

*Read at a meeting of the Chicago Laryngological and Otological Society, December 5, 1932.

were involved early, followed by the third, fourth, seventh and eighth. He ran the gamut of symptoms from the cranial nerves from paresthesia and analgesia of the face and neck to blindness, immovable eyeball, proptosis, tinnitus, deafness, vertigo, loss of smell and partial paralysis of some of the facial muscles. All of the marked symptoms occurred in the last six months of his illness. During the years of 1928, 1929, 1930 and the first half of 1931 he attended to business almost every day, was in general good health and did not seem greatly depressed.

On the whole, this patient presents the rather remarkable picture of living in comparative comfort for four or five years with an inoperable carcinoma of the epipharynx which was apparently kept in control for many months by massive doses of radium and X-ray.

AUTOPSY REPORT.

January 13, 1932. T. R., male, age 40. An infiltration tumor mass which in some locations is pinkish gray, firm and fibrous (cartilaginoid consistency) and in other places shows areas of yellow granular necrosis, occupies a large area in the left middle fossa. The dura mater covering the medial half of the middle fossa has been largely replaced by firm tissue which on the inside has invaded the left temporal lobe of the cerebrum, the posterior part of the left frontal lobe, the left side of the pons, the left optic tract and the optic chiasm. The left optic and oculomotor nerves are completely surrounded by the tumor and apparently replaced by it. There is an extension of the tumor into the left optic foramen with the posterior third of the orbit filled with soft friable pink tissue. On the outside of the dura the floor of the middle fossa is eroded in many places and in several different parts; both tables of the skull have been broken through. The internal auditory canal is filled with tumor tissue and segments of the seventh and eighth nerves and the Gasserian ganglion apparently have been completely destroyed. Medially the body of the sphenoid has been penetrated and the hypophysis invaded from the left side. The posterior ethmoid cells on the left side are filled with pink friable tumor tissue. The sphenoid sinus has been obliterated completely. The bony tissue between the posterior part of the internal nares and epipharynx below and the region of the sella turcica and medial wall of the middle fossa above is so soft and thin that much of it can be removed with a knife. From the roof and left wall of the nasopharynx a lobulated firm tumor projects into the lumen. It is attached by a broad base to the wall of the pharynx and spreads in all directions along the mucosa as processes with raised indurated edges. The surface is ulcerated, granular and hemorrhagic in several places. The intervening tissue, including bone between the epipharyngeal tumor and the mass within the cranial cavity is almost completely replaced by hyperemic tumor tissue which is firm in some places and soft and friable in others. Metastases in other parts of the body except the cervical glands on both sides, were not found.

Microscopic Findings.—Sections from the tumor tissue in the vault of the pharynx show an abrupt transition from stratified columnar epithelium of the normal mucosa to the thick masses of atypical epithelial cells which constitute most of the tumor masses. The tumor cells are large, polyhedral to thick spindle shaped with large round or oval hyperchromatic

nuclei and one or two distinct, sometimes large, dark nucleoli. They are arranged in thick mantles around branching connective tissue stalks. The connective tissue is moderately vascular and in many places is composed of young connective tissue cells. Large areas of necrosis are found in those areas which are farthest separated from the vascular connective tissue.

Sections from the firmer portion of the tumor (the mass found at the base of the brain) show a relatively much greater proportion of fibrous matrix so that in some places solid plugs and stellate masses of epithelium are isolated in the dense connective tissue.

The portions of the tumor which project into cavities of rarified bone and into the nasal sinuses show numerous small papilliform processes on the free surface, each with a small central core of connective tissue.

The hypophysis shows a normal histologic structure except in those portions which have been invaded by the tumor cells. This invasion, however, has not been extensive except on the extreme left of the anterior lobe.

Microscopic Diagnosis.—Transitional cell carcinoma (arising from nasopharyngeal mucosa), with extensive infiltration of the base of the skull, meninges and brain.

COMMENT.

A search of the literature* fails to reveal a previous authentic case of involvement of the eighth nerve by direct extension from a carcinoma of the nasopharynx.

Ball[1] reports a case of carcinoma of the nasopharynx in which during the course of the disease a nonfunctioning labyrinth occurred which he attributed to involvement of the eighth nerve at the internal acoustic meatus. No autopsy was reported.

A few cases of symptoms from the eighth nerve due to indirect pressure from the nearby tumor have been described.[2]

Several cases of metastatic carcinoma of the eighth nerve[3 4 5 6 7] were found, usually secondary to carcinoma of the lung.

Waltman,[8] in 1922, compiled forty-four cases from the literature of malignant tumors of the nasopharynx with involvement of the nervous system, and reports twenty-five cases of his own seen at the Mayo Clinic. No case of direct eighth nerve involvement from carcinoma was found. Two cases of sarcoma of the nose or nasopharynx with eighth nerve involvement are listed. One case was reported in 1867 by Verneuil and Schweich. The other was reported in 1874 by Veillon.

*Acknowledgment is made to Miss Marian Kappes of the Webster Library of the Evanston Hospital for her valuable help in the search of the literature, especially in the foreign journals.

Gatewood,[9] in 1916, found only one case of carcinoma of the nasopharynx with metastasis to the viscera. This case was a small metastasis to one kidney.

MARSHALL FIELD ANNEX BLDG.

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LXIX.

A CASE OF RHINOSCLEROMA IN A NATIVE BORN AMERICAN.*

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ANN ARBOR.

Although rhinoscleroma is a disease encountered heretofore chiefly in central, eastern and southern Europe, it now seems probable that it is a disease which is practically world wide. We now find that cases have been reported from such varying geographical locations as the United States, Cuba, Central and South America, Japan, China and Sumatra.

About forty cases have been described in this country in foreign born patients, the majority being from Russia, Poland and Austria.[1]

In addition to these, we find in the literature four cases of rhinoscleroma in American born patients. The first report was made by Wende,[2] in 1896, at which time he did not report the microscopic, pathologic or bacteriologic findings. The second reported case is that of a sailor who visited Central and South America. The findings in this patient were published by Watkins in 1921.[3] The third case was treated by Wood in 1925,[4] and the fourth came under the observation of Figi and Thompson at the Mayo Clinic in 1927.[5]

The case herein recorded is the first of rhinoscleroma to be observed in the University of Michigan Clinic in more than 300,000 admissions.

The patient, whose parents were born in Poland, was herself born in Michigan. She had never been away from the vicinity of her birth-place until she entered the hospital, and none of her relatives or acquaintances are known to suffer from this malady.

A. S., a white, single American female of seventeen, one of a family of seven children, born in Michigan of parents born in Poland, came to the University of Michigan Hospital August 31, 1932. She complained of

*Reported from the Department of Otolaryngology, University of Michigan Hospital.

severe dyspnea with especial difficulty during inspiration. The present illness started five months previously, at which time she had entered another hospital and had been delivered of a full term still-born fetus without incident. Following the delivery her dyspnea improved, and no further investigation was made of her respiratory trouble. During the five months previous to admission, the dyspnea increased, accompanied by periods during which she could talk only in a whisper. At times she expectorated small amounts of thick tenacious greenish yellow sputum, which was difficult to raise. Recently her breathing had become noisy during both inspiration and expiration. She had lost twenty-five pounds in weight, and during the previous two weeks had vomited frequently.

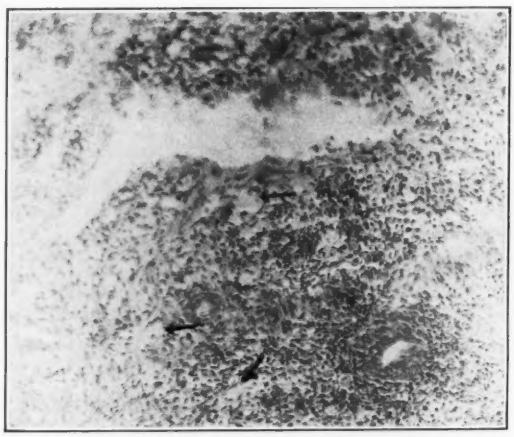


Fig. 1. Photomicrograph (low power) showing round cell infiltration and masses of foam (Mikulicz) cells. Foam cells are represented by the light staining areas between the nuclei of the round cells (indicated by the arrows).

Examination revealed a fairly well developed white adult with anxious expression, and obvious respiratory obstruction in the upper trachea or larynx. With each respiration there was a harsh rasping wheeze, although breathing seemed easier with the head turned to the left.

The chest showed a flare of the ribs more marked on the right than the left. Auscultation demonstrated a transmission of the harsh wheeze throughout the chest with no other abnormal lung findings. The heart sounds were normal, and the abdomen and extremities were negative.

The nose was normal externally, but within the nasal cavities was a mass of crusting purulent discharge with some irregularity of the mucosa. This was considered, upon first examination, to be due to a chronic sinus

disease, and not till much of the fetid discharge was removed, could it be determined that the pathologic process consisted of granulation tissue resembling that seen in a chronic infective granuloma.

Laryngeal examination showed good approximation of the vocal cords, but their vibratory motion was interfered with by a smooth subglottic annular constriction of the laryngeal lumen, impinging particularly from the posterior aspect. This constriction extended downward, diminishing the size of the tracheal lumen to about one-eighth of its normal caliber.

Roentgenograms of the larynx showed the infiltration circumscribing the larynx and trachea, but somewhat more extensively on the posterior wall. A direct laryngeal examination was performed with a view to

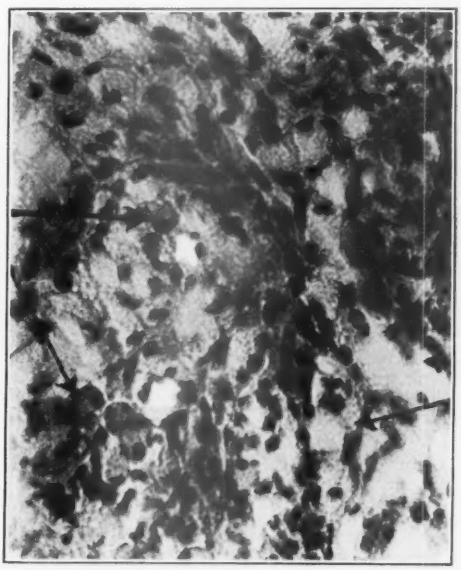
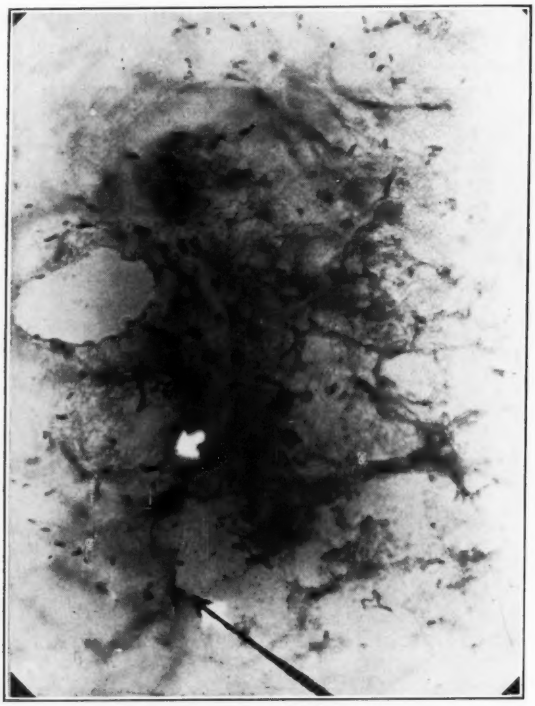


Fig. 2. Photomicrograph (high power) showing foam (Mikulicz) cells and hyaloid (Unna) bodies.

obtaining material for microscopic study, but this procedure greatly increased the dyspnea and necessitated immediate tracheotomy. The trachea was opened through the third, fourth and fifth tracheal rings. At this level there was no thickening on the anterior wall, although the posterior wall was elevated and roughened, probably indicating the presence of the lesion at that point.

Following tracheotomy a series of biopsies were performed, but on each occasion no more than mucosa was removed so that the true identity of the pathologic change could not be determined. With a fourth attempt

enough tissue was obtained to show the typical infiltrating granulomatous changes characteristic of rhinoscleroma, including the Unna bodies, the Mikulicz cells, and by special stains the short diplobacilli of Frisch. Biopsies from the inferior turbinates and the nasal septum showed the same lesion. Cultures from the nasal mucosa yielded bountiful growths of the short encapsulated, non-motile, Gram-negative diplobacillus, which on a blood agar medium produced a viscid colony, making gas and acid



Frisch bacilli.

Fig. 3. Photomicrograph (oil emersion) showing organisms (Frisch bacilli) in tissue (silver stain).

in dextrose medium, but none in lactose medium. With these clinical, pathologic and bacteriologic observations a definite diagnosis of rhinoscleroma was made. (Pathologic specimens studied and reported by Dr. C. V. Weller.)

During the patient's stay in the hospital, general therapeutic measures were instituted and large doses of potassium iodide were administered.

This merely lessened the amount of sputum and decreased the crusting in the nose, the drug seeming to have no favorable effect upon the lesion. An autogenous vaccine was prepared from the nasal culture and thirty-nine subcutaneous injections ranging from 0.1 cc. to 1.0 cc. were given. This seemed to improve the condition of the larynx sufficiently to make possible the removal of the tracheal tube, but after thirty-six hours respiration again became difficult and the tube was replaced. Deep X-ray therapy was then instituted. Seven portals of entry, four over the larynx, and three over the nose, were used. Through each portal 400r units were given on seven of eight successive days. Six days after the treatment was completed, examination of the larynx showed a marked improvement. The tracheal tube was corked and the patient breathed satisfactorily through the larynx. Several days later the tracheal tube was again removed and the tracheal fistula closed spontaneously while the laryngeal and tracheal lumen remained widely open, permitting normal breathing even during exertion. The nasal lesions greatly improved, the mucosa assumed a normal appearance, and the crusting had entirely disappeared when the patient was discharged from the hospital.

COMMENT.

Of the three specific kinds of treatment, namely, potassium iodide, auto-vaccination and radiotherapy, the latter was the only one which had any real effect. In a discussion of this case with Dr. C. V. Weller, he mentioned a very logical explanation for the efficacy of the X-ray. Three stages of this disease have been described by a number of foreign authors. First, a catarrhal edematous stage, in which the tissue is identical with any tissue undergoing an inflammatory reaction. In this stage a definite diagnosis of rhinoscleroma cannot be made. Specimens from our first biopsies in the above case were obtained from tissue in this stage. Second, the characteristic granulomatous tissue stage, in which the Mikulicz cell, the Unna bodies and Frisch bacilli appear and produce the greatly increased size of the lesion. Specimens from the nose and deeper portions of the subglottic lesion were typical. Third, a final stage of fibrosis in which the granulomatous tissue is replaced by firm contracting scar.

The effect, then, of the radiation therapy, is to hasten the third stage. The decrease in the size of the lesion, as seen by indirect laryngoscopy, by the roentgenograms and by rhinoscopy, was probably due to a fibrosis resulting from the radiation. Hence it may be pertinent to state that the sooner this form of treatment can be administered the more rapid will be the evolution of the

lesion to its final stage, and any evidence of recurrence should be treated in like manner.

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LXX.

CASE OF PARAGEUSIA ASSOCIATED WITH CHRONIC SUPPURATIVE OTITIS MEDIA.

LANCELOT BARNES, M. B.,

TORONTO.

The writer was interested in a paper by Mr. M. Vlasto of London, published in the *Journal of Laryngology and Otology* for January, 1930, dealing with lesions of the chorda tympani, under the title "The Chorda Tympani Nerve in Otology." Mr. Vlasto discussed chiefly ageusia and sublingual and submaxillary gland pathological findings, as a result of tympanic operations, mentioning also the possibility and occurrence of parageusia with chronic suppurative otitis media. In the discussion which followed one of his colleagues mentioned one such case (parageusia). From the scanty literature on the subject the present writer believes these cases to be rather infrequent.

The history is as follows:

J. L., dental surgeon, married, aged 38, on September 29, 1931, complained of:

1. Dull intermittent feeling of pressure in the right side of the head for several years.

2. Discharge (occasionally foul) from the right ear for ten years. Never profuse and sometimes absent for several weeks at a time.

3. Peculiar foul taste in the right side of the mouth for about five years.

During the past two years has had several attacks of coronary artery disease (confirmed by electrocardiograph). Some months ago had lower right first molar extracted (because some root changes showed in an X-ray), thinking the taste must be due to this. Thinks his breath must be foul smelling and wonders if his patients smell it. (His nurse has never noticed any foul breath.) Wonders if there is a sinus at site of extraction to account for taste. The patient can touch the exact spot of the peculiar dirty taste, located adjacent to the site of extraction on the side of the tongue and floor of the mouth. It is invariably in the same place.

Examination: Nose and throat negative; tonsils previously removed; right ear, large sized perforation in posterior portion of drum with pus in auditory canal.

The possibility of a lesion of the chorda tympani causing the taste was discussed with the patient. X-ray of the lower jaw was negative. X-ray of the right mastoid showed considerable sclerosis. Alcohol instillations were prescribed pro tem.

January 14, 1932. No discharge from right ear today. Foul taste somewhat relieved lately.

July 13, 1932. Ear discharging moderately again. Patient's physician anxious to have every possible source of infection eliminated for the sake of the heart condition.

August 18, 1932. Right radical mastoidectomy.

September 29, 1932. Mastoid cavity completely epithelized and dry. Since operation foul taste has been conspicuously absent, with only a "slight suggestion" very infrequently. No headache.

December 8, 1932. Cavity dry. Says he rarely thinks about the taste at all now. Only occasionally is reminded of it.

Discussion of this case with several colleagues brought up the possibility that the foul taste might be due to discharge from the eustachian tube. (This possibility was also mentioned by Mr. Vlasto in a personal communication to the writer.) It is difficult to reconcile this idea, however, with the fact that the foul taste was invariably described in the same small region, and further disproof is shown by the fact that infrequent returns of the taste occur in the presence of a dry middle ear (existent since the radical operation).

48 Bloor St. E.

LXXI.

NASOCILIARY NEURALGIA CAUSED BY OSTEOMA.*

RALPH A. FENTON, M. D.,

PORTLAND, ORE.

Successful solution of a complex diagnostic problem gives particular satisfaction when the individual concerned has become a psychic risk in addition to his physical discomforts.

This case report concerns M. L., a handsome girl of 20, seen first in March, 1932, because of violent bilateral low frontal and temporal headache, slightly worse on the right side, made worse by studying, and relieved somewhat by athletics or other outdoor occupation. Thirteen years before she had accidentally injured the right orbito-ethmoid region when she collided with a pile of tiling at night, a jagged fragment piercing the inner part of the right orbit. She had since been subject to minor headaches about once a week, with slight dizziness but no vomiting. Recently, drowsiness had accompanied her headaches; there had been slight giddiness on change of posture during two previous months, without sense of rotation or falling. She also made vague complaints of 'things going black before her eyes" and of "difficulty in getting her breath, on rising in the morning." There was slight postnasal discharge, but no complaint of colds or nasal obstruction. She had been obliged to drop out of school, and social activities annoyed her; the family complained of her irritability and sensitiveness, marking a total change in a disposition formerly most cheerful.

Physical examination disclosed slight serous discharge from the sphenoethmoidal region on both sides, with slight lateral pharyngitis. Lipiodol entered these cavities, but could not be injected or drawn by suction into either frontal sinus. All the sinuses seemed clear roentgenologically and on transillumination. A small dense shadow, typically that of an osteoma, appeared in the right anterior ethmoid region. Blocking either the sphenopalatine or the anterior ethmoid region seemed to dull the headache considerably, but it returned with greater violence after the local anesthetic had worn off. Marked tenderness was found over the anterior surface of the right frontal, also just below the orbital rim. A small diagonal linear scar over the right nasal bone marked the site of her old injury.

Vision in the right eye was 20/70, left 20/30, inadequately corrected by lenses fitted the preceding year. The eyes were externally normal save for a slight weakness of the right internus, causing diplopia in extreme abduction. The discs were normal except for a slight elevation of the

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*From the Department of Otolaryngology, University of Oregon Medical School. Read before the Pacific Coast Oto-Ophthalmological Society, San Francisco, June 30, 1933.

nasal margin in the right eye, without vascular change. The fields were normal. There was no nystagmus.

Vision was brought up to 20/20 by

O. D. +0.37 sph ⌒ +1.25 cyl ax 90°
O. S. +0.25 sph ⌒ +0.37 cyl ax 180°,

and exercise prisms, O. U. 1½ △ base out were used with some relief for about a month, when aggravation of the mental symptoms suggested neurologic consultation.

Dr. A. J. McLean found a repeated positive left Babinski on the outer side of the foot, negative however from the inner side; and a rather weakened right knee jerk. Slight broadening of the sellar roentgenogram he considered within normal bounds, and he felt that ventriculography was not then indicated. His diagnosis lay between hypophysial tumor, orbito-ethmoid osteoma, or meningioma. Under his management, with prescription of a combination of amidopyrin and phenobarbital, she remained in comparative comfort for several months.

Permanent prescription of

O. D. +1.00 cyl ax 90° ⌒ 2 △ B O
O. S. +0.25 cyl ax 180° ⌒ 1 △ B O

was made four months later, and seven months later than her first examination, both vision and muscle balance remained normal and comfortable under the new correction. But the headaches persisted, and a melancholic attitude toward family and friends forced a decision to undertake operative relief.

Eliminating intracranial neoplasms, since no field, fundus or pulse disturbances had developed, there remained the question of spheno-ethmoid surgery, of alcohol injection of the anterior ethmoidal nerve, and of removal of the little osteoma. The sinuses had remained clear, hence were not attacked; and the difficulties and dangers of anterior ethmoidal alcohol injection removed this procedure from serious consideration.

The osteoma, which lay apparently in an anterior ethmoid cell, was sought under general anesthesia November 26, 1932, but was located just behind the old scar in the extreme lower part of the frontal sinus, totally occluding the nasofrontal duct. The first chisel cut showed it glistening through the normal sinus mucosa, attached mesially by a narrow vertical synarthrosis. The sinus was quite dry and healing by first intention followed three days in the hospital. Her headaches and other symptoms disappeared at once; the young woman returned within a month to her schooling, has since regained ten pounds, and her family and friends testify to her entirely normal temperament and disposition.

Most osteomata, even of very great size, are painless.[1] Chance placed this one across some filaments of the nasociliary, now called (BNA) the anterior ethmoid nerve. Sluder[2] points out that this nerve, which enters the orbit between the heads of the external

rectus and the divisions of the third nerve, passing inward over the optic nerve to the inner side of the orbit below the superior rectus and oblique, leaves the orbit by the anterior internal orbital canal, furnishing branches to the ciliary ganglion, long ciliary branches to the eyeball, and an infratrochlear branch. Within the cranial cavity it grooves the outer edge of the cribriform plate, reaching the ethmoid sulcus and descending to the nasal fossa, where it supplies the septum and follows a groove in the nasal bone to the skin of the bridge and tip. Branches are also described to the superior and internal recti, to the levator, and offsets also reach the frontal sinus and anterior ethmoid cells.

Sluder's differential diagnosis[3] from frontal sinusitis, tenderness over Ewing's point, was not possible in this case, owing to the external scar. He cites only three or four cases of this neuralgia, all more or less acute, irregular and spasmodic, but complaining as did this girl of the pressure of glasses on the bridge of the nose. None of his cases arose from osteomatous pressure.

Barnhill[4] mentions the pressure of exostoses from the petrous against the gasserian ganglion as a cause of trigeminal neuralgia, approving Frazier's dictum that minor neuralgias of this type are usually from the sphenopalatine distribution. Frazier's classification[5] includes (1) Typical major trigeminal neuralgias; (2) trigeminal pressure or scar neuralgias, from neoplasms or following herpes zoster; (3) toxic neuralgias, mainly ophthalmic, from digestive and other sources; the probable sympathetic system neuralgias, paroxysmal in the orbit, temple, cheek or neck, occasionally with generalized headache or hemicrania, and (5) the psychalgias.

In the presence of frightful headaches, with muscular imbalance, hyperopic astigmatism at opposite axes, slight chronic posterior ethmoiditis, a suspicious looking sella turcica with unequal lower limb reflexes, dizziness and marked psychic changes, it required much prayerful cogitation before risking opening a clean frontal, on the chance that removal of a pea-sized osteoma would settle the whole problem. One may say at least that the end justified the means.

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LXXII.

SEQUESTRATION OF THE COCHLEA: REPORT OF TWO CASES.*

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AND

WILLIAM B. CHAMBERLIN, M. D.,

CLEVELAND.

The literature shows that sequestration of parts of the temporal bone is most frequently found in cases in which mastoiditis is a complication of scarlet fever, but one may also find the condition in any case of chronic mastoiditis. Typically the patient is conscious only of a chronic discharge from the ear. This discharge has a fetid odor and there is a progressive loss of hearing. These manifestations are but casually noticed until suddenly acute symptoms arise with such severity that the individual is incapacitated for work and must seek a doctor's advice. By this time most of the irreparable damage has been done, and any operative procedure is an emergency one dealing with serious complications.

All patients with chronically discharging ears are potentially poor health risks unless the cavities from which the discharge comes are accessible for frequent cleansing and inspection. If, following radical mastoid operations, there are remaining areas of moisture in the cavity, frequent inspection should be made for underlying pathologic changes. Since so much information concerning petrositis has come to light recently, we can remain no longer so calm as formerly when moisture appears to be coming from the mouth of the eustachian tube, because it may be coming from a sinus tract leading to infection deep within the pyramid.

Recurring granulation tissue within the mastoid cavity or the middle ear should always be examined for its possible cause. The most common causes of such tissue are remaining bone chips or

*From the Division of Otolaryngology of the Department of Surgery of the Lakeside Hospital and the Western Reserve University School of Medicine.

sequestra. Of course, there is always some granulation tissue to deal with during the healing of a radical mastoid cavity, but under the usual treatment it readily dries up and covers over with epithelium. Fibrous bands or false membranes may conceal deeper necrosis. It is only when the entire cavity is covered by a smooth and dry epithelial lining that one can be assured that the patient is free from chronic infection with its sequelæ.

Any case of necrosis of a part of the inner ear must necessarily cause deafness with an associated loss of vestibular function. As Ingersoll[1] reports, compensation may be complete in as little as four and a half months. Usually the case of sequestration of the labyrinth is also complicated by a paralysis of the seventh nerve. This is accounted for by the close relationship of the sequestrum to the horizontal or vertical portion of the fallopian canal.

A painstaking history should elicit symptoms of dizziness and possibly nausea during the course of the disease. The history of such symptoms was obtained from our patients only after much questioning, because the duration of the symptoms was for one week only and was explained in the patient's own mind as being "flu." There was a small epidemic of influenza prevalent at the time.

Politzer[2] says that when the sequestrum has been discharged or removed, the cavity thus formed is filled with granulations which ossify after being transformed into fibrous connective tissue (callus). In other cases the walls of the cavity become covered with scar tissue which, through the invasion of the epidermis of the external meatus, is changed into the matrix of a cholesteatoma. During the healing process cavities often form. Frequently exostoses and hyperostoses obliterate the defect or there is narrowing and obliteration of parts of the tympanic cavity and meatus. Scar tissue may cause an atresia of the external canal. There is always a high degree of disturbance of hearing or there may be total deafness.

The two cases reported here illustrate how much necrosis of bone may take place from infections which the patients consider lightly because they cause so little inconvenience. And yet the process is serious enough to endanger their lives. In fact, the

disease is so insidious as to pass unnoticed by the surgeon for a long period of time.

Case 1.—N. Z., a male patient, aged 36, first came to the outpatient department of Lakeside Hospital on September 2, 1931. He had had discharge from both ears for two years, with gradually diminishing hearing in both. There had been no pain until two weeks previous, when he had begun to have severe pain in the left side of the head associated with dizziness. Examination at this time revealed a bilateral purulent aural discharge with large polypi in both canals. There was also mastoid tenderness on the left side, with complete loss of hearing in this ear and only one foot of hearing for conversational voice in the right ear. Upon looking to the right or to the left there was a horizontal nystagmus to the side towards which the eyes were turned. With the head inclined thirty degrees forward there was a horizontal nystagmus lasting for twenty seconds when the patient was turned either to the right or to the left.

The patient was admitted to Lakeside Hospital, where a left mastoidectomy was performed on September 3, 1931. The mastoid was sclerotic throughout with a large cholesteatoma in the region of the antrum. This had eroded some of the neighboring bone. The usual radical mastoid operation was performed on the left side. The polyp was removed from the right canal. The postoperative course was entirely uneventful, and the patient returned home on the eighth postoperative day.

Further postoperative care was carried out in the outpatient department. This consisted in removing the granulation tissue which recurred persistently and in cleansing the cavity of the foul discharge which accumulated. The patient returned for treatment at intervals of three to five days over a period of five months. Throughout this time he would have periods of severe pain in the left side of the head, radiating down the neck and lasting from an hour to several days. Because of the severity of the pain the patient was again admitted to the hospital on February 10, 1932, but was discharged after five days, when the pain subsided without operative interference. Two days later, in the outpatient department, a bony sequestrum of the cochlea was located with a probe and easily removed with forceps (Fig. 1). The pain did not recur until April 19th, when the patient became uncomfortable and large granulations were found throughout the region of the middle ear cavity and a portion of the tegmen. Because of the pain a secondary radical mastoidectomy was performed on April 21, 1932.

During the operation no evidence of the horizontal semicircular canal or any of the two verticals could be seen, although the facial nerve lay exposed in its vertical portion. In spite of this there was no facial paralysis. The postoperative course was uneventful and the patient was discharged from the hospital on the ninth postoperative day free from all pain. Since the operation there has been no recurrence of pain or granulation tissue, and the cavity has become completely covered with epithelium.

The final examination before the patient was discharged from outpatient care revealed a completely deaf ear on the left with a dead labyrinth on the same side. At no time had there been any signs of involvement of the seventh nerve.

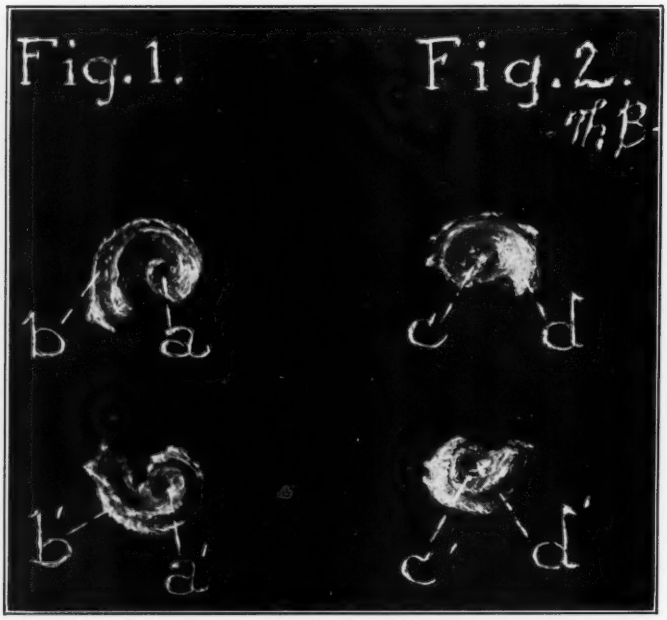

Fig. 1—Case I. Fig. 2—Case II. Sequestra of cochlea. *a* and *c* are under side of apex (cupula); *á* and *ć* show apex viewed from above; *b* and *b'* show one and one-half turn of spiral; *d* and *d'* about one turn of spiral.

Case 2.—N. C. was a colored male of 19 years, who had had a discharging right ear for eighteen years. He was admitted to Lakeside Hospital on August 24, 1931. For two weeks prior to admission there had been continuous severe headache but no dizziness.

Examination was negative except for the right ear, the canal of which was filled with foul, purulent material and a large polyp obscuring any possible view of the drum or middle ear. There was marked tenderness to pressure over the mastoid tip. Conversational voice could be heard at contact only. The left ear and drum membrane were negative. There was a spontaneous rotatory nystagmus with the quick component to the right.

At the time of the radical mastoidectomy on August 25, 1931, a large cholesteâtoma was found in the middle ear and antrum. Some necrosis of bone was found over the facial nerve. A portion of the dura and lateral sinus was uncovered during the operation. Otherwise the usual radical mastoidectomy was performed.

The postoperative course was uneventful with the temperature normal by the sixth day. The patient was discharged on the tenth postoperative

day. Granulations which repeatedly sprang up were removed under local anesthesia in the outpatient department. The cavity became extremely sensitive to touch and filled with very dense granulation tissue. At the same time the patient complained of so much pain that it was necessary to remove the granulating mass under gas and oxygen anesthesia on November 13, 1931. Following this operation the granulations were removed at intervals of two to seven days in the outpatient department, but they always recurred. In addition, a stenosis of the external auditory meatus was gradually forming so that it was necessary for the patient to be readmitted to the hospital for a secondary radical mastoidectomy.

The findings at this operation, on January 21, 1932, disclosed large cholesteatoma with granulations in the middle ear and along the exposed dura. These were removed and a secondary plastic operation was performed to enlarge the external meatus. The postoperative course was again uneventful and the patient was discharged on the sixteenth hospital day.

During the removal of granulations in the outpatient department on February 24, 1932, an acute inflammatory condition was observed in the right ear. This was manifested by pain in the ear, redness around the postauricular incision and increased discharge from the cavity. On March 8, 1932, during the routine cleansing of the mastoid cavity, a sequestrum of a portion of the cochlea was removed (Fig. 2). Following this the pain disappeared, the granulations disappeared, and the discharge became less until March 18, 1932, when the cavity was found to be healed and free from pus or granulations. The patient has been entirely free from pain since that time.

The final examination for release from the dispensary revealed deafness in the right ear with complete loss of labyrinthine function on this side. At no time had there been any signs of involvement of the seventh nerve.

CONCLUSIONS.

1. Sequestration of the cochlea need not necessarily have scarlet fever as an etiologic factor.

2. Sequestra may separate from the cochlea without any acute symptoms.

3. Sequestra of the inner ear do not always cause paralysis of the seventh nerve.

4. If granulations continue to develop in the cavity following a radical mastoidectomy, careful examination should be made for underlying sequestra.

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LXXIII.

BRONCHO-ESOPHAGEAL FISTULA: REPORT OF CASE.*

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This case is of interest because of the rarity of the condition, the duration of the symptoms, observation of a sign which can be considered as suggestive if not diagnostic, the findings at esophagoscopy and by the use of a radiopaque substance in the fistula and the method of treatment.

The patient, a woman, aged 23 years, noticed that for the past seven years there was cough associated with swallowing of food. This was not constant. Cough was more marked when taking liquids or highly seasoned foods. On one occasion a very violent paroxysm of coughing followed taking a dose of aromatic spirits of ammonia and the patient was conscious of a sense of suffocation.

Roentgen studies of the swallowing function by Dr. G. M. Nelson and Dr. J. T. Farrell indicated the presence of a fistulous communication between the esophagus and a branch of the left lower bronchus. After the ingestion of barium it could be seen in the respiratory passages at a distance from the esophagus.

The Ono sign observed in a case of tracheo-esophageal fistula† was elicited by Dr. Jo Ono in this case. His examination was as follows: "The patient was placed successively lying flat on the back, on the left side, the right side, sitting upright and sitting and leaning forward. In each of these positions patient was given sips of warm milk to swallow. From seven to ten seconds after the act of swallowing, the patient was seized with a violent coughing attack. The expectorant consisted largely of the fluid which had just been swallowed. The attacks of cough were more violent with the patient lying on her left side, sitting upright and leaning forward than in the other positions. Ausculatory examination with a stethoscope placed at the sixth interspace, left paravertebral line, revealed a few moist rales. However, shortly after patient took a sip of water there

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*Presented at the meeting of the American Bronchoscopic Society at Washington, D. C., May 10, 1933.

From the Department of Bronchoscopy and Esophagoscopy, Jefferson Medical College Hospital, Philadelphia, Pa.

†Bucher, C. J., and Ono, J.: Tracheo-esophageal Fistula of Syphilitic Origin: Report of a Case. Proceedings of College of Physicians of Philadelphia, 1932.

were heard numerous medium and coarse moist rales, and this was followed by sudden coughing attacks. Following the coughing attacks only a few rales could be heard over the same area."

At esophagoscopy there was found a small depression about 1 cm. in depth in the left wall of the esophagus. At the bottom of this was observed a small fistulous communication about 3 mm. in diameter (Fig. 1).

Fig. 1. An esophagoscopic image of the small funnel-like depression observed on the lateral wall of the esophagus. The mucosa appeared smooth, not unlike that observed in the esophagus. The septum between the esophagus and orifice of the fistula was thin and resembled a bronchial spur.

Fig. 2. Roentgenograms made with the esophagoscope introduced in the esophagus to the level of the fistula and aspirating tubes placed to indicate the position of the fistula and esophagus. In *A*, a straight flexible tipped tube has been passed into the esophagus beyond the end of the esophagoscope. A curved flexible tipped tube, introduced into the openirg of the fistula has pursued a direction to the left and posterior. In *B*, a small quantity of iodized oil, 40 per cent, introduced into the fistula through the curved tube, has passed up- and outward. In *C*, the oil has distributed itself in subdivisions of the bronchus of the lower lobe. (Films by Dr. W. F. Manges.)

During inspiration the opening was readily visible, whereas during expiration and cough it collapsed and a small quantity of pus was extruded. At a subsequent examination air bubbles could be seen at the opening during expiration. With the patient holding her breath and bearing down, a sound produced by air being forced through the fistula was distinctly heard by those in the operating room. The opening was 29 cm. from the upper teeth.

With the aid of Dr. W. F. Manges, additional studies were carried out (Fig. 2). These corroborated the earlier studies and conclusively demonstrated that a communication existed with the lower lobe of the left lung.

In considering the plan of treatment to be employed in obliterating the fistula it was decided to destroy the mucous membrane by topical applications of silver nitrate. In the event that this failed, surgery was to be employed. Silver nitrate, 30 per cent, was applied to the esophageal end of the fistula with a resultant narrowing but not obliteration of the opening. A silver nitrate stick was then applied to the orifice on a special carrier. A Rehfuss tube was inserted for feeding purposes. When observed esophagoscopically several weeks later the orifice of the fistula appeared to have been obliterated.

128 South 10th St.

LXXIV.

PERICARDITIS AS A TERMINAL LESION IN MEDIASTINITIS: REPORT OF A CASE, FOLLOWING THE INHALATION OF A FOREIGN BODY IN THE BRONCHUS AND ACCOMPANIED BY SPONTANEOUS EMPHYSEMA.*

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The usual cause of death given following a diagnosed lesion in the mediastinum is acute mediastinitis.

This would seem to be but a contributing cause. The close connection of the mediastinal lymphatics and the pericardium could easily lead to a pericarditis. The case report with autopsy would seem to prove this contention.

P. M., 2¼ years old, was given a handful of peanuts, while his parents were not watching him. This occurred on March 27, 1932. He had a severe attack of coughing, and since then has had severe paroxysms of coughing. On March 29th it was noticed by his parents that there was a swelling under the angles of both jaws. By March 30th the infant's neck and chest were swollen and seemed to get worse after a paroxysm of coughing. Temperature has ranged from 100 to 105 degrees for the past two days.

On admission, March 30, 1932, to the babies' ward of the New York Post-Graduate Hospital, a tentative diagnosis was made of a foreign body in the right bronchus, pneumonia secondary to the foreign body and subcutaneous emphysema.

The patient was seen a few hours after admission, and there was a subcutaneous emphysema of the scalp, face, neck and chest, extending to the second rib anteriorly and sixth dorsal vertebræ.

Physical examination of the lungs showed diminished breath sounds over the right lower lobe and crepitant rales of the emphysema heard everywhere.

The heart was normal in size and there was no displacement.

Roentgenograms showed a foreign body pneumonia in the right lower lung, probably upper mediastinitis and subcutaneous emphysema.

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*Presented at the meeting of the American Bronchoscopic Society at Washington, D. C., May 10, 1933.

From the Laryngological Service of the New York Post-Graduate Medical School and Hospital, Columbia University.

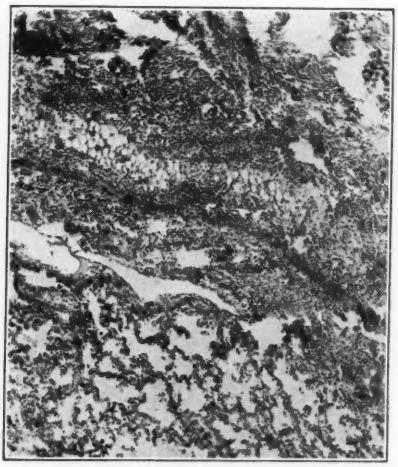

Fig. 1. There is an acute purulent infiltration of the pleura and the mediastinum. Lung infiltrated.

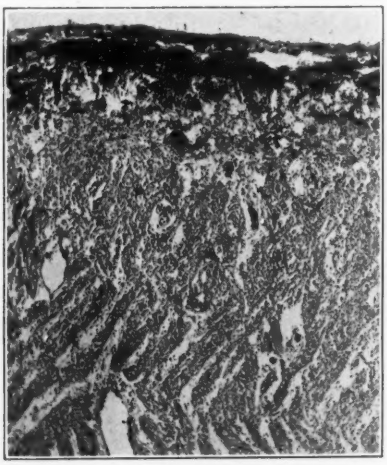

Fig. 2. Pericardium shows edema, fibroblastic proliferation and infiltration with leucocytes, mononuclear cells and polymorphonuclear lymphocytes. The surface is covered by a thick layer of purulent exudate consisting of a large amount of fibrin, in the meshes of which a large amount of debris, red blood cells and polymorphonuclear cells are present. Edema and cellular infiltration extends into the underlying myocardium for a short distance

Fig. 3. Area between the bronchus and the aorta, showing area of necrosis.

Fig. 4. Area of necrosis between the aorta and esophagus. Enlarged lymph nodes.

A bronchoscopy was done and a large piece of peanut was removed from the right middle lobe bronchus and a smaller piece from the right lower lobe bronchus. Several smaller particles were aspirated from the right lower lobe bronchus.

The left bronchi contained no foreign bodies.

For the next two days the child seemed to improve, but the subcutaneous emphysema remained about the same, and for the next five days he seemed to be holding his own, but the temperature was irregular and there were occasionally paroxysms of coughing. Roentgenograms showed a central pneumonia and physical signs confirmed this.

It was felt that there might possibly be a piece of peanut still in the lung, and another bronchoscopy was done but no foreign body was seen. The mucosa of the right main, middle and lower lobe bronchi was very much congested and edematous.

The subcutaneous emphysema lessened but never disappeared entirely.

For several days the child was given oxygen and was transfused twice. However, on April 11th he died.

Several roentgenograms, taken April 1st, 5th and 7th, showed a central pneumonia of the right lung. The postmortem examination showed:

Acute purulent pericarditis with effusion.

Acute purulent superior mediastinitis.

Cloudy swelling of the kidney and liver.

Subacute splenitis.

No foreign bodies in bronchi.

The explanation of the subcutaneous emphysema was never satisfactorily accounted for. No break could be found in the tracheal or bronchial mucosa at postmortem. However, the superior mediastinum showed a large amount of thick, creamy pus, and this also was seen along the vessels and along the course of the trachea. There must have been a break in the mucosa in the party wall between the trachea and esophagus, and this healed after the first few days following the inhalation of the peanut. There never was an increase in the emphysema, although it did not disappear entirely.

The extension from the mediastinum to the pericardium through the lymphatics produced the terminal lesion that was the main cause of death.

Roentgenograms and physical findings did not definitely show the pericarditis.

108 East 38th St.

LXXV.

TRACHEOBRONCHIAL DIPHTHERIA; TRACHEOTOMY; REPEATED BRONCHOSCOPIC REMOVAL OF MEMBRANES: RECOVERY.*

ETHAN F. BUTLER, M. D.,

ELMIRA, N. Y.

The patient was a female white child, 6 years of age, who had enjoyed average good health until her present illness. Four days before admission to the Arnot-Ogden Memorial Hospital she became acutely sick with diphtheria. The correct diagnosis was not made at home (a small town in Northern Pennsylvania, some forty miles from Elmira), and not until the child was brought to Elmira on account of increasing severity of symptoms was the nature of the disease recognized or any appropriate treatment instituted.

At time of admission her temperature was 100, pulse rate 78, respirations 28. The child was of an ashy gray color. Her respirations were stertorous and labored, and the respiratory excursion was limited. There was inspiratory retraction of all interspaces. Breath sounds were diminished. Her tonsils were enlarged but no membranes were apparent. In the depths of the hypopharynx there could be seen a membrane. Direct laryngoscopic examination showed an intensely inflamed larynx. The vocal cords were still mobile, but the airway was greatly diminished. Thick, tenacious secretions were present in the trachea. Immediate tracheal aspiration afforded great relief. Diphtheria antitoxin was administered in massive doses—30,000 units intravenously and 40,000 units intramuscularly.

The relief from tracheal aspiration was only temporary. Within four hours of the initial aspirations there had been several recurring attacks of intense dyspnea and cyanosis. Tracheotomy was, therefore, performed under local anesthesia. For thirty-six hours there was little respiratory embarrassment, the routine removal of secretions sufficing for the child's needs. Thirty-six hours after tracheotomy, however, there was extreme respiratory difficulty. The child became cyanosed and respirations labored. The breath sounds were fairly well transmitted on the left side, but were greatly diminished on the right. It was impossible to relieve the condition by aspiration through the tracheotomy tube. Therefore, a 5 mm. bronchoscope was introduced through the tracheotomy wound, disclosing thick, dark colored, tenacious secretions. Even so, the effort to remove all these secretions were not entirely successful, but an adequate airway was reestablished with immediate improvement in the child's condition.

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*Presented at the meeting of the American Bronchoscopic Society at Washington, D. C., May 10, 1933.

From the Chest Clinic, Arnot-Ogden Memorial Hospital, Elmira, N. Y.

Four hours later the child was again in desperate condition. A second bronchoscopic effort was rewarded by the removal of a thick, putty-like brown tracheal cast. For twelve hours the child appeared to be safe, then there was a sudden recurrence of asphyxial symptoms and a third bronchoscopic exploration was carried out. No casts were obtained, but a large quantity of thick secretion was removed. Again, six hours later, there was another sudden attack of intense dyspnea and a second tracheal cast was removed by bronchoscopic means. After another six-hour interval labored breathing demanded a fifth bronchoscopic exploration. The secretions appeared to be less tenacious and more fluid at this time, justifying hope that further instrumentation would be unnecessary and, indeed, for eleven hours the condition of the child was satisfactory. At the end of that interval, however, extreme dyspnea recurred and the sixth bronchoscopic exploration resulted in the removal of a softer, lighter colored tracheobronchial cast which had formed in the trachea and left main bronchus. From that time the condition of the child showed steady improvement. However, a seventh and last precautionary bronchoscopy was performed after a lapse of eleven hours. One small plug was removed.

Convalescence from this point progressed favorably. Nine days after the tracheotomy a smaller tube (No. 2) was substituted. Four days later the child began to talk and a No. 1 tube was substituted for the No. 2. Fifteen days after tracheotomy the tube was permanently removed. The wound rapidly closed. The patient was finally discharged from hospital twenty-two days after admission, the opening into the trachea being completely closed and the wound reduced to a small granulating area. Final examination showed a practically normal larynx.

As is usual with such cases, there was evidence of both myocardial and renal irritation. These associated conditions, however, never caused serious alarm.

Two months after discharge from hospital and again eight months after discharge the child was brought in for physical examination and fluoroscopy. Neither laryngoscopic nor bronchoscopic examinations were done on these subsequent visits. In all respects the progress of the child was found to be satisfactory.

Comment.—This case is presented in order to demonstrate an ultimate satisfactory result following persistent and frequently repeated efforts to keep the airways free of obstruction. Tracheal aspiration, without visual control, would not have sufficed in this case. Tracheotomy alone would not have prevented asphyxia due to the casts which formed and reformed rapidly in the neighborhood of the bifurcation. It was only by visual control and the ability to securely and accurately grasp the casts that success was finally obtained. The possibility of entering the trachea directly through the tracheotomy wound was a helpful factor. Not only did it permit the use of a larger tube, but it also saved

the patient's larynx from seven bronchoscopic attacks within a period of fifty-two hours.

The massive doses of antitoxin contributed undoubtedly to the child's ultimate recovery. They did not, however, prevent the formation of four tracheal or tracheobronchial casts within the next ensuing five days.

370 W. Church St.

LXXVI.

PROTECTING LENS FOR THE BRONCHOSCOPIST'S SPECTACLES.*

SAMUEL IGLAUER, M. D.,

CINCINNATI.

In order to obviate the frequent changing of spectacles which become soiled during a bronchoscopy, the lens shown in the accompanying illustration (Fig. 1) has been devised to protect the bronchoscopist's eyeglasses. The device consists of a single fit-

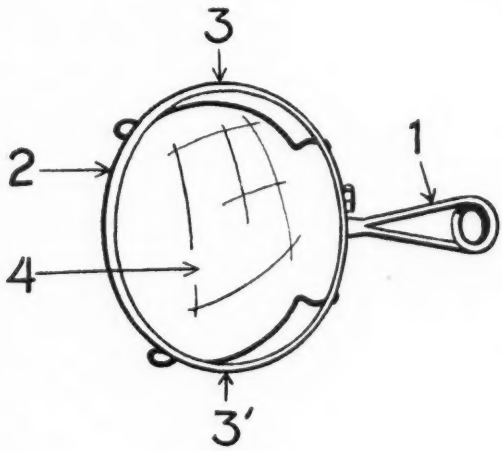

FIGURE 1.
1. Handle (2.5 cm.)
2. Frame
3-3'. Spring
4. Lens (4.2 cm.)

over frame containing a plano-meniscus lens. The fit-over frame slides over the spectacle mounting corresponding to the eye which the bronchoscopist is using. When the protecting lens becomes

*Presented at the meeting of the American Bronchoscopic Society at Washington, D. C., May 10, 1933.

soiled by sputum, a nurse can remove the frame and lens and, after cleansing the same in hot water, can slide it back onto the operator's spectacles. The heat imparted to the glass will for a time prevent fogging of the glass by the patient's breath. If the operator prefers, a second frame and lens can be held in reserve for an immediate change. The device can be constructed by any optician.

707 RACE STREET.

LXXVII.

PERFORATION OF THE LARYNX BY A METALLIC FOREIGN BODY REMOVED BY LARYNGOFISSURE.*

SAMUEL IGLAUER, M. D.,

CINCINNATI.

On December 24, 1932, a mechanic, aged 30 years, while pounding a pinion gear with a heavy hammer, felt a piece of steel strike him in the neck, causing him to gasp for a moment. A gush of blood issued from his neck, and shortly thereafter he heard a gurgling sound in his throat and began expectorating considerable blood. He was brought to the Cincinnati General Hospital immediately after the accident.

On admission, the patient continued expectorating bright red blood at frequent intervals, bringing up from 2 to 3 cc. each time.

Examination: There was a small, dry, puncture wound about one-half cm. long slightly to the left of the midline over the lower border of the thyroid cartilage. No fullness of the neck was noted, but subcutaneous crepitation could be elicited on both sides of the midline. Marked tenderness was present over the larynx and upper trachea anteriorly. The voice was not impaired. Mirror examination showed the presence of fresh blood in the larynx. A few coarse bronchial or tracheal rales could be heard over the chest.

In the roentgenograms a small, dense foreign body could be seen in front of the body of the seventh cervical vertebra. (Fig. 1.) In the lateral view the foreign body appeared to be imbedded in the tracheo-esophageal wall. (Fig. 2.)

From the examination and X-ray findings it seemed apparent that the blood was continuously trickling into the trachea through a perforation. The X-ray picture indicated that the foreign body was imbedded in the party wall between the trachea and esophagus, from which position it did not seem likely that it could be removed either by tracheoscopy or esophagoscopy. I therefore determined upon an external operation to control the bleeding and to remove the foreign body.

About three hours after the accident, under novocain infiltration, a vertical incision was made with its center including the puncture wound in the neck. The trajectory of the foreign body could easily be followed

*Read before the meeting of the American Bronchoscopic Society, at Washington, D. C., May 10, 1933.

Fig. 1. A small metallic foreign body is seen in the midline of the neck on a level with the body of the seventh cervical vertebra. (There is also evidence of subcutaneous emphysema in the neck.—Dr. McGehee.)

Insert: The piece of steel removed from the larynx.

through the soft tissues. There was profuse bleeding in the region of the cricoid cartilage. This was controlled by clamps and ligatures. A small perforation was then seen in the lower part of the thyroid cartilage, just to the left of the midline. A median incision was made through the first tracheal ring and cricoid cartilage, and the incision was extended upward to include the lower two-thirds of the thyroid cartilage. Upon retracting the cartilages, a small lacerated wound was found in the mucosa covering the posterior surface of the cricoid cartilage. At first the foreign body could not be seen, but upon making pressure alongside of the lacerated mucous membrane the foreign body was extruded and removed. The laryngostomy incision was closed with catgut sutures passed through the perichondrium, and the incisions in the soft parts of the neck were closed with silk sutures. The foreign body was a very sharp, flat piece of steel measuring 6 x 4 mm. (See Insert Fig. 1.)

There was practically no bleeding after the operation, and the patient made an uninterrupted recovery. He was discharged from the hospital on the sixth day, and by the tenth day he had a normal voice.

Fig. 2. In the lateral position the foreign body appears to be imbedded in the tracheo-esophageal wall.

Comment.—An external operation was performed upon the patient to control hemoptysis due to blood trickling into the trachea through a perforating wound. The X-ray findings indicated that the foreign body was imbedded in the tracheo-esophageal wall, so that its removal by either tracheoscopy or bronchoscopy was deemed impossible. It is, of course, possible that the foreign body might have been removed by the attraction of an electromagnet introduced into the larynx. It is rather remarkable that the blow of a hammer imparted sufficient momentum to the piece of steel to cause it to penetrate the larynx.

707 RACE STREET.

Abstracts of Current Articles.

NOSE.

Changes in the Nasal Accessory Sinuses After Birth.

Wasson, W. Walter, M. D. (Denver), Arch. of Otolaryng., 17:197 (February), 1933.

This is a preliminary report and no definite conclusions are drawn, but the author believes that aeration of the nasal accessory sinuses in the newborn infant occurs slowly.

Physiology of these cavities is not understood during the first two months of life, but probably effects them in later life. X-ray diagnosis is difficult at this age. TOBEY.

Effects of Radiation on Allergic Nasal Mucosa: A Further Report.

Bernheimer, L. B., M. D., and Cutler, Max, M. D. (Chicago), Arch. of Otolaryng., 17:658 (May), 1933.

In the Archives of October, 1932, forty cases were reported where radiation had been carried out on hyperesthetic rhinitis. The clinical results after one year's time are reported here. Fifty-two per cent of the patients irradiated were symptom free after one year. Twenty per cent were relieved of nasal obstruction and watery discharge. Eight per cent had improved breathing space, 10 per cent no relief, and 10 per cent very slightly relieved.

A good discussion of this subject follows the paper. TOBEY.

Clinicopathologic Study of Tumors of the Nose, Pharynx and Mouth of Teratologic Origin.

Furstenberg, A. C. (Ann Arbor, Mich.), Trans. Am. Laryng. Assoc., 1933.

A clinical study of tumors of teratologic origin found in the nose, pharynx and mouth was made. A microscopic diagnosis of these neoplasms was recorded, the characteristics of the cells were described, and the degree of malignancy was considered. Attention was given to an embryologic and pathologic study of unusual neoplasms found in the upper respiratory tract. Reference was made to the therapeutic measures employed in the treatment of these lesions. Surgical intervention and roentgenotherapy were considered. IMPERATORI.

Experimental Surgery of the Nose and Sinuses. III. Results Following Partial and Complete Removal of the Lining Mucous Membrane from the Frontal Sinus of the Dog.

Hilding, Anderson, M. D. (Duluth, Minn.), Arch. of Otolaryng., 17:760 (June), 1933.

This paper deals with two sets of experiments (1) where the entire mucous membrane was removed from ten frontal sinuses of eight dogs; (2) large portions of the mucous membrane were removed from four sinuses of four dogs. The operative technic is given and the results in detail.

The author concludes that after removal of the lining membrane from the normal frontal sinus of dogs, the sinus usually fills with scar tissue, but in exceptional cases partial restitution of the sinus with regeneration of epithelium takes place. In some cases the cavity remains with thick connective tissue lining it and epithelium may or may not cover this.

If portions of epithelium are left in the sinus, cysts form in the scar tissue. TOBEY.

The Nasal Accessory Sinuses: Some Recollections and Reflections.

Tilley, Herbert (London), Journal of Laryng. and Otol., 47:808 (December), 1932.

An interesting address was given by the author, in which he kindly gave some advice and encouragement to the younger members of the profession and spoke on the development of the modern methods in diagnosing and treating nasal sinusitis.

In chronic maxillary sinusitis the author prefers the canine fossa operation, as a general rule, to the intranasal route, because it seems only rational to expose a hidden pathologic condition to view and to examine its nature and extent. An exception, of course, is made in favor of the intranasal route in children and young adults with good teeth and without any particular complication.

Woakes, in 1885, first associated nasal polypus with inflammatory lesions of the structures composing the ethmoid bone, and to Hajek we are indebted for his pioneer work on this subject. The importance of a detailed knowledge of the anatomy and pathology of the ethmoid labyrinth is emphasized. It is the "key situation" of the nasal sinuses, and command of this position is

the most important factor in a successful attack upon inflammation in any or all of them.

The sphenoid sinuses are generally involved when extensive ethmoid inflammation is present, but sometimes this sinus is the only one involved. Pain over the mastoid is not an infrequent symptom of chronic sphenoid sinus suppuration.

In frontal sinus operations the dreaded complication of osteomyelitis may be due to the exposure of the diploe by a too energetic use of the curette in the removal of an inflamed and septic mucous membrane or to deficient postoperative drainage. The author advises a conservative attitude in most cases of frontal sinusitis. The endonasal operation will in many cases relieve the symptoms, and in a fewer number it will bring about a permanent cure. However, fatal osteomyelitis has been recorded in a few instances following the endonasal operation.

The mature laryngologist will read with pleasure Mr. Tilley's resumé of his ripe experiences in nasal sinus surgery, and will especially approve the sound conservatism advised. Those who have just started as specialists in rhinology should read and reread this paper, for it will save them from many bitter disappointments and some of their patients from unnecessary surgery and maybe disasters. GOLDSMITH AND MCLEOD.

Minute Anatomy of the Sphenoid Ostium.

Proctz, Arthur W. (St. Louis), Trans. Am. Laryng. Assoc., 1933.

Although the minute structure of the nasal mucosa and that of the sinuses has been often described, no attention has been given to their actual junction at the ostium. This study was made chiefly for the purpose of determining the relations of the various tissues to the edge of the bone. It was thought that this relation might be such that a vascular engorgement might cause a nerve impaction to which headaches could be traced. Such is not the case. The thin fibrous lining of the sinuses stops abruptly near the edge of the bone, and the ostium is lined definitely by nasal mucosa. There are no structures which cross the bony edge, but all fibers lie parallel to one another, at the extreme membranous margin of the ostium. The direction of the fibers is such that swelling is primarily toward the nose and not toward the center of the ostium. IMPERATORI.

Experimental Surgery of the Nose and Sinuses.

Hilding, Anderson, Ph. D., M. D. (Rochester, Minn.), Arch. of Otolaryng., 17:321 (March), 1933.

These experiments were carried out on dogs with normal sinuses, and it was found that ridges and diaphragms follow the removal of strips of mucous membrane on concave surfaces and these interfere seriously with drainage. The removal of a small circular strip of mucous membrane is likely to result in two separate chambers of a sinus, but this can be avoided by having the cut edges closely approximated without leaving any bare bone.

An ostium can frequently be closed by taking a circular piece of mucous membrane from around it and leaving bare bone.

TOBEY.

The Influence of Septic Infection of the Sphenoid Sinus upon the Cerebral Blood Supply.

Pickworth, F. A. (Birmingham), Journal of Laryng. and Otol., 47:797 (December), 1932.

Research work is presented, together with evidences from the literature, to show that the cerebral arteries may be adversely affected by contiguous sepsis of the sphenoid sinus and nasopharynx. The mechanisms by which this takes place are discussed under the headings of diffusion of toxic substances, centrifugal spread of organisms, and irregular constriction or dilatation of cerebral vessels due to involvement of the sympathetic system.

Cases illustrating the association of diseased sphenoid sinuses with the pathologic changes of the internal carotid artery are given. Organisms from the sinuses involving the perivascular tissues of the carotid artery have been demonstrated. Involvement of the pituitary gland occurs not infrequently from infection of the sphenoid sinuses. Probably the most usual path of subacute infection by organisms occurs by retrograde thrombosis of veins. Nasopharyngeal sepsis may affect the cerebral arteries through disturbance of the sympathetic system, either by involvement of the nasal ganglion or by the cerebral branches arising from the main sympathetic trunk in the neck. The article is illustrated by several unusually fine colored drawings.

GOLDSMITH AND MCLEOD.

PHARYNX.

Two Unusual Cases of Tumors of the Neck with Involvement of the Pharynx.

Patterson, Norman (London), Journal of Laryng. and Otol., 48:49 (January), 1933.

Case 1.—Cervical Cyst Involving the Larynx and Pharynx.

Patient was a man, aged 46 years, who during three months had had three recurrences of a swelling of the right side of the neck, and each time it had been aspirated or excised. When examined by the writer the swelling occupied the upper part of the right side of the neck, extending from the level of the angle of the jaw down to the center of the anterior border of the sternomastoid. The swelling extended forward across the middle line, and it seemed to be attached to the thyroid cartilage. It was tense and appeared to be cystic in character. A large swelling was present on the right side of the pharynx at the level of the upper aperture of the larynx. The right side of the epiglottis was pushed over to the left so as to hide from view a portion of the larynx.

At operation the cyst was excised and there was considerable difficulty as the capsule was ill defined. The cyst wall was very adherent to the thyroid membrane and lateral wall of the pharynx, and it was only with extreme care that perforation of the pharyngeal wall was avoided. There is now an entire absence of both external and internal swellings, and the patient does not complain of any symptoms.

The wall of the cyst resembled a thyroglossal cyst, but the latter cannot cause a swelling as described above. Illustration of the case was given.

Case 2.—Branchial Cyst Undergoing Malignant Degeneration.

The patient was a man, aged 54 years, who had a swelling on the right side of the neck of three months' duration. It rapidly increased in size and at examination was about the size of half a small orange. Definite fluctuation was present and the skin was not adherent to the tumor. There was a condition of the buccal mucous membrane resembling leukoplakia with some superficial ulceration. Vincent's organisms were isolated. In the larynx on the right side in the region of the aryepiglottic fold there was a

smooth swelling, evidently produced by the tumor in the neck. The condition was considered to be probably a branchial cyst.

The tumor was explored at operation and found to be adherent to the surrounding structures. The cyst was freely excised and found to be covered in part by hard nodular excrescences. It was impossible to remove the cyst wall and the wound was partially closed.

The pathologist considered the case to be one of branchial cyst which had undergone malignant degeneration.

The case was treated with radon seeds around the growth. Two and a half months later the swelling in the neck had greatly diminished and the mouth had improved. But a condition resembling leucoplakia was still present. The swelling on the right side of the larynx had increased in size.

Case 3.—Retropharyngeal Lipoma.

Patient was a man, aged 42 years, who in February, 1928, was first noticed to have a swelling behind the right tonsil. He was treated with X-rays under the impression that he was suffering from a malignant growth. The cervical glands were said to be enlarged. When examined by the author there was a large tumor, which occupied the greater portion of the right side of the pharynx. It extended beyond the middle line and passed up behind the soft palate, displacing this structure and pushing the right tonsil markedly forward. It extended downwards into the laryngopharynx. The swelling was soft and did not give the impression of lobulation. Underneath the upper part of the right sternomastoid there was an indefinite swelling, which became much more marked on deglutition. The swelling was diagnosed as a soft tumor of innocent type, probably a lipoma.

At operation the tumor was exposed by an incision in the neck. It was very adherent, due probably to the previous treatment. Removal of the tumor was accomplished. However, the patient developed delirium tremens and infected the wound by removing the dressing. He died two weeks later from bronchopneumonia. Histologically the tumor was proved to be a lipoma.

The case is of special interest on account of the mistaken diagnosis, which led to errors in treatment and greatly increased the difficulty of the operation. GOLDSMITH AND MCLEOD.

The Tonsil Problem.

Kahler, Otto (Freiburg), Journal of Laryng. and Otol., 48:2 (January), 1933.

The author discusses the physiologic rôle of the tonsils and the so-called "chronic tonsillitis," particularly in its relation to general affections. The tonsils belong to the lympho-epithelial system and must be considered as an integral part of the total mesopharyngeal mucous membrane. The lymphatic tissue distributed in the rest of the pharyngeal and intestinal mucous membrane performs the same function as the tonsillar tissue. The hypotheses which consider the tonsils acting as a protective mechanism or functioning as endocrine glands are discussed. Although very little is known about the function of the tonsils, they form a very small part of the lymphadenoid tissue of the oral cavity and pharynx. The author believes that complete removal of the tonsils does not do any harm and this is confirmed by clinical experience.

The pathologic findings in chronic tonsillitis and their relation to other diseases, such as rheumatic diseases, endocarditis, septic diseases and nephritis, are discussed. The observations that were made are illustrated by photomicrographs. Tonsillectomy is greatly preferred to any other method of treatment in chronic tonsillitis. GOLDSMITH AND MCLEOD.

Focal Infection of Tonsillar and Dental Origin (Fokalinfektiöse Symptome tonsillären und dentalen Ursprungs; bei "rheumatischen" Muskel und Gelenkleiden).

Gording, Dr. R., and Bjorn-Hansen, H. (Oslo), Acta. Otolar. Suppl., Oslo, 1933.

This study of 171 pages is based on exhaustive blood counts and metabolic tests on 137 patients, to decide what symptoms of a general and rheumatic nature may be ascribed to such infective sources as teeth and tonsils, and also how far the Arneth leucocytic shift to the left may be trusted as a diagnostic means of recognizing such focal infective changes.

Removal of foci, with subsequent observation during three years, was carried out on 64 persons. Of this number, 52 with an average age of 31, became free of clinical signs and were considerably improved. The blood picture showed infection in 46,

returning to normal in 43, while three cases with pulmonary symptoms remained positive.

Twelve persons formed the second, or "acute rheumatic" group, average age 39, and had a history of acute articular rheumatism. Seven of the eight who were improved after removal of teeth or tonsils showed normal blood at the end; one with lung involvement did not show the improved blood picture. Recurrences were frequent in this group, four out of eight; but only two out of fifty-two in the focal infective group, which gave a history of long standing arthritic changes without acute rheumatic fever.

Rather an exact correspondence between leucocytosis above the normal of 6,500 along with increased shifting to the left, and failure to secure relief after removal of foci, was observed in both classes of patients. Complete protocols of sixty-four cases are given. The monograph is excellent, carefully written, and presents an accurate analysis of accurately recorded data. A five-page English summary is included. FENTON.

Immediate Tonsillectomy for Peritonsillar Abscess (La amigdalectomia immediata en el tratamiento de los abscesos paratonsilares).

Zambrini, Prof. A. R., and Padesta, Dr. R. (Buenos Aires), Rev. Argent. de O. R. L., 1:755 (December), 1932.

One hundred cases of peritonsillar abscess treated by immediate snare tonsillectomy under local anesthesia healed very rapidly with no greater average of bleeding than in a similar number of cases done under ordinary conditions. FENTON.

Pyemia Following Acute Tonsillar Infections.

Rubin, Herman, M. D. (Brooklyn), Arch. of Otolaryng., 17:183 (February), 1933.

A review of the literature is given on this subject. It is not a rare condition and occurs in from 1 to 2 per cent of acute tonsillar infections.

Phlebitis may extend downward and involve the internal jugular and facial veins or upwards to reach the cavernous sinus by way of the pterygoid plexus or petrosal sinuses.

Writers disagree as to the route of infection, whether by phlebitis or lymph vessels.

The anaerobic streptococcus is the organism most frequently found and blood cultures are positive in 50 per cent of cases.

The Tobey-Ayer test is recommended in diagnosis. In treatment, the para-pharyngeal space should be exposed and ligation with resection of any thrombosed veins.

Four cases are reported here. TOBEY.

Treatment of Tonsillar Sarcomas by Deep X-Ray Therapy (Tratamiento de las amigitalas sarcomatosas por la radioterapia profunda).

Tapia, Dr. M. G. (Madrid), Rev. Esp. Amer. de L. O. R., 23:653 (November), 1932.

Calling attention to the practical inoperability of tonsillar sarcomas when diagnosed (operative mortality 80 to 90 per cent), Tapia reports ten cases, nine of whom are clinically well after two years, with disappearance of the growth. He advises massive doses, repeated three or four times several days apart, and cervical or supraclavicular glands must also be irradiated powerfully. FENTON.

LARYNX.

Giant Tuberculoma of the Larynx: Six Year Cure by Laryngofissure (Tuberculoma Laringeo Gigante—Emilaringectomia Guarigione da Sei Anni).

Malan, A. (Turin), Arch. Ital. di Otol., Rino. e. Laryng., 44:15 (January), 1933.

The author reports the case of a woman, aged 56, farmer, complaining of difficulty in talking and dyspnea on exertion.

Laryngoscopy demonstrated that the left half of the laryngeal vestibule was occupied by a large mass, smooth, light red in color, covered by an intact mucosa, crossed by three large vessels. By probe pressure he could feel an elastic sensation. There were no enlarged glands palpable in the cervical region.

Laryngofissure was performed by the Glück technic under novocain anesthesia. The tumor, being encapsulated, was easily enucleated.

Histologic examination of the neoplasm demonstrated the formation of large nodules of typical tubercular structure (although no Koch bacilli were found), giving the typical picture described by Hajek—that is, that tuberculoma is a submucus infiltration of circumscribed type resembling a tumor. No trace of pulmonary involvement was found after exhaustive examinations of all types. SCIARRETTA.

Acute Streptococcal Ulceration of the Epiglottis.

Tause, H. B. (Nottingham), Journal of Laryng. and Otol., 47:762 (November), 1932.

Three cases of phagedenic ulceration of the epiglottis, the ages ranging from 30 to 45 years, are reported. The outstanding features of the cases were the sudden onset with rapid development of serious symptoms, and the agonizing pain on swallowing, although the pharynx presented a comparatively innocent appearance. There was profound depression and toxemia. The ulcers were confined to the anterior surface of the epiglottis, and covered by greenish-yellow slough. Swabs showed a predominance of hemolytic streptococci but were negative to diphtheria and Vincent's organisms.

The outstanding symptoms of pain, mental distress and thirst were relieved only by hypodermics of morphia and rectal salines. Antistreptococcal serum was given in each case. The author believes that quicker and better results might be obtained if a large initial dose of antiscarlatina serum was given, followed by a full dose daily till the temperature falls.

GOLDSMITH AND MCLEOD.

Malignant Diseases of the Larynx and Pharynx.

Stewart-Harrison, R. (Zurich), Journal of Laryng. and Otol., 47:725 (November), 1933.

The treatment of malignant disease of the larynx and pharynx by the protracted-fractional method of deep X-ray and the results of 144 cases are reviewed. The importance of the histologic appearances in relation to the sensitivity of the tumor is shown and illustrated by photomicrographs. The principles involved in this method of deep X-ray therapy and details of the technic are given. By lengthening the time of irradiation, by decreasing the size and increasing the number of the single treatments, it is possible to obtain a different effect between essential and malignant tissues, by which the malignant tissues will be more effectively destroyed and the healthy tissue will suffer no irreparable damage. The absolute necessity of carrying the irradiation to the stage of a severe reaction is emphasized. By accurate dosage the author has never seen serious sequelæ as a result of protracted-fractional radiation. Illustrative examples showing variation from case to case are given.

The results of the treatment since its introduction to Zurich in 1929 are analyzed on a basis of the situation, nature and extent of the various growths. The oldest case was treated only three and a half years ago and the word "cure" has been avoided, but the cases which have become free from signs and symptoms as a result of the treatment and up to the present time have not developed a local or regional recurrence are discussed. The author believes that the results of protracted-fractional X-ray therapy in the treatment of cancer of the larynx and pharynx are not inferior and are often superior to those obtained by any other treatment. GOLDSMITH AND MCLEOD.

Cysts of the Larynx.

Myerson, M. C. (New York), Trans. Am. Laryng. Assoc., 1933.

Congenital cyst of the larynx is probably of more frequent occurrence than is supposed. The condition is seen in infancy and early childhood and is mistaken for and treated for diphtheria, papilloma, congenital stridor and, in fact, any condition in which the laryngeal lumen is obstructed. No doubt some cases of asphyxia of the newborn and obscure deaths in infancy are due to this condition. In one of the cases reported the patient had a cyst of the tracheal wall in addition to the cyst in the larynx. The methods of treatment are discussed. IMPERATORI.

Speech Movements in Action.

Stetson, R. H. (Oberlin, O.), Trans. Am. Laryng. Assoc., 1933.

The essential movements of speech occur above and below the larynx; this is apparent in the use of the artificial larynx and in whispering. The larynx sets the pitch, but the fundamental movement in speech is a pulse of the breathing muscles which makes the syllable in speech and the note in singing. The consonant figures in releasing and arresting this breath pulse. The author described the function of the intercostals and of the abdominal muscles in speech, two types of esophageal speech and the muscular blocks of stammering. Slides were presented showing evidence of the above (1) from subglottal changes in air pressure in subjects with a tracheotomy tube and in persons using an artificial larynx, (2) from kymograph tracings of speech movements and (3) from action current records of the concurrent contractions of the groups of muscles involved in speech. IMPERATORI.

Multinodular Origin of Cancer of the Vocal Cords (Sur l'origine multicentrale du cancer de la corde vocale).

Benjamins, Prof. C. E. (Groningen), Rev. de Lar., 54:274 (March), 1933.

Discussing cancers of a cord which shows swelling of the entire cord when first seen, Benjamins reports two cases of squamous cell carcinoma which disclosed microscopically several distinct nodules separated by pachydermatous areas. He ascribes this development at multiple centers to extension of the chronic irritant all along the cord, like multiple epitheliomata from tar. Hemilaryngectomy with local anesthesia has kept the patients free from recurrence for over a year. FENTON.

Contact Ulcer of the Larynx: Report of a Case.

Imperatori, Charles J. (New York), Trans. Am. Laryng. Assoc., 1933.

Contact ulcer of the larynx is a name given by Chevalier Jackson to superficial ulceration occurring on one or both sides of the larynx posteriorly. The lesion may be bilateral, and is usually located at the vocal process and posterior to it. Usually the lesions are of some chronicity. A case with lesions lasting over one and one-half years is reported. The last pathologic report shows the beginning of some atypical cell proliferation. What the ultimate outcome in this patient will be cannot definitely be foretold. A report will be made at some future date. IMPERATORI.

Primary Tuberculosis of the Laryngopharynx (Tubercolosi Neoproduttiva Faringo-Laringea).

Torrini, U. L. (Florence), Arch. Ital. di Otol., Rino. e. Laryng., 44:24 (January), 1933.

The author reports this case only because it is firmly believed that tuberculous lesions in the nasopharynx and larynx are always secondary to pulmonary tuberculosis, although it has been reported previously that tuberculous lesions have been found in the adenoid tissue of children apparently healthy.

This patient, a child 6 years of age, had the nasopharynx completely filled with a tumor mass, forcing the soft palate forward. The epiglottis, aryepiglottic folds and laryngeal ventricle were completely filled with it, causing difficult respiration and making it impossible to see the rima-glottis. Blood Wassermann was negative, tuberculin cutaneous reaction strongly positive, but ex-

haustive investigations did not reveal tuberculosis anywhere else. Tracheotomy was performed to prevent suffocation.

The histologic examination of the tissue removed gave the typical picture of tuberculosis. The two microphotographs accompanying this article prove this clearly. SCIARRETTA.

EAR.

Problems Concerned with Empyema of the Petrous Apex.

Kopetsky, Samuel J., M. D. (New York), Arch. of Otolaryng., 18:47 (July), 1933.

The author feels that there has been a great deal of confusion as regards infection of the petrous pyramid and he tries here to clarify the subject. He distinguishes between the cellular or pneumatic pyramid and the sclerotic or noncellular, as these two types become infected.

The literature is reviewed and the various operative procedures as advocated by Eagleton, Voss, Freckner, Ramadier, Almour and Kopetsky are discussed.

The author's operation is used only in the pneumatic type of infected pyramid. TOBEY.

The Genetic Factor in Otosclerosis.

Davenport, Milles and Frink (New York), Arch. of Otolaryng., 17:503 (April), 1933.

These authors conclude that roughly 0.2 per cent of the white population of the United States is otosclerotic. Deafness was noticed first at from 4 to 55 years of age.

The original data were obtained by house to house visits of trained eugenic workers who gave auditory tests, etc. Sixty new families were studied to get at the law of inheritance.

Approximately twice as many females as males were found affected. Otosclerotic females seem more slender. When both parents are affected nearly all daughters are otosclerotic and two-thirds of the sons. When only the mother has otosclerosis the affected sons and daughters are equal.

When only the father is affected, the daughters are affected 50 per cent more than the sons. He feels that otosclerotics do not belong to a degenerative class and have a different genetic basis from deafmutism or labyrinthine hardness of hearing.

Many family trees are shown diagrammatically. TOBEY.

Cervical Rheumatism Causing Vertigo (El reumatismo cervical conin causa de estados vertiginosos).

Jiménez-Díaz, Prof. C. (Madrid), Rev. Esp. Amer. de L. O. R., 23:697 (December), 1932.

Numerous cases of so-called rheumatism of the neck, including nodular myositis especially affecting the trapezius, often associated with shoulder and occipital pain, were found to present entirely normal hearing and vestibular responses; but these people complained bitterly of dizziness on movement. The author considers that hypersensitiveness of the nerve endings in these muscles determines a diminished labyrinthine irritability of the affected side, and cites experiments now in progress wherein chilling the skin of the neck along the sternomastoid, below the ear, gives rise to definite nystagmus. FENTON.

Latent Heredity Syphilitic Labyrinthitis (Labirintite Eredo-Luetica Tardiva).

Carnevale-Ricci, F. (Milan), Arch. Ital. di Otol., Rino. e. Laryng., 44:65 (February), 1933.

The author discusses in detail latent heredity syphilitic labyrinthitis and reports his observation in two cases of young children (ages 9 and 14, respectively). In the first case the lesion developed gradually, first in one ear and at a later period in the other, with alternations of improvement and aggravation, until there was complete bilateral deafness. In the second case the affliction developed suddenly in both ears, while the patient was in perfect health, and in a few days deafness became complete.

He describes in detail the pictures of latent heredity syphilitic labyrinthitis and brings out the importance of various symptoms based on the two cases reported. SCIARRETTA.

Surgical Treatment of Otosclerosis (Le traitement chirurgical de l'otospongiose).

Sourdille, Prof. M. (Nantes), Rev. de Lar. 53: (Sept.-Oct.), 1933. (Supplementary pamphlet.)

This lecture, given in French and English, presents full details and pictures of Sourdille's combined atticotympanotomy, partial ossiculectomy, plastic closure of the aditus and attic, and final fistulization of the horizontal semicircular canal with establishment of a membranous flap over the new opening. FENTON.

Role Which the Epidermis Plays in Suppurations of the Middle Ear.

Druss, Joseph G., M. D. (New York), Arch. of Otolaryng., 17:484 (April), 1933.

The author gives an excellent review of some of the knowledge and theories on this subject. He has studied 120 temporal bones in serial section; thirty-eight of these cases had a suppurative ear. The repair of acute perforations and some of the factors associated with chronic suppuration are discussed. Theories of primary cholesteatoma are mentioned. He mentions the fact that not only patients with suppuration of the middle ear but those who have been apparently cured, with or without operation, should be kept under observation. TOBEY.

Otitis Media in Scarlet Fever.

Williams, Horace J., M. D. (Philadelphia), Arch. of Otolaryng., 17:235 (February), 1933.

In a series of 14,733 patients with scarlet fever, otitis media occurred in 10.8 per cent. The incidence of surgical mastoiditis was 1.1 per cent.

Paracentesis when indicated tended to lower the incidence of surgical intervention on the mastoid.

Aural complications may arise at any stage of scarlet fever.

There were only seven cases of extension to the meninges in 14,733 cases of scarlet fever and two cases of lateral sinus thrombosis.

The author has not observed the rapid destruction of the ear drum so often mentioned in texts, and he waited for signs of mastoiditis before operating.

The average length of time for recovery following operation was 47.8 days. TOBEY.

Vidian Neuralgia with Special Reference to the Eye and Orbital Pain in Suppuration of the Petrous Pyramid.

Vail, Harris H., M. D. (Cincinnati), Arch. of Otolaryng., 17:212 (February), 1933.

Anatomic studies show the close relation between the great superficial petrosal nerve and the interior portion of the petrous pyramid.

It is also recognized that impulses can pass along the great petrosal nerve and, by way of the sphenopalatine ganglion, anastomose with the terminal branches of the ophthalmic division.

Three cases are reported to show that irritation of the vidian nerve in the floor of the sphenoid sinus causes orbital pain.

The gasserian ganglion and its branches are above the dura and therefore the dura must be involved to affect them.

It appears to the author that the ocular and orbital pains so typical of petrous suppuration are not due to irritation of the ophthalmic division of the fifth nerve but to irritation of the great petrosal nerve caused by suppuration in the petrous bone.

TOBEY.

Complications of Otitis Media Without Rupture of the Membrana Tympani.

Love, Andrew A., M. D. (Los Angeles), Arch. of Otolaryng., 17:297 (March), 1933.

The author states that this subject has frequently been discussed, but no one has stressed the occurrence of intracranial complications.

Nine cases are reported in detail and in each one a complication was present, ranging from perisinus abscess to more serious trouble.

Love found hemolytic streptococcus more frequently and considers it the most dangerous bacterial agent. Children were more frequently affected than adults.

Swimming was the cause of this type of mastoiditis in many cases.

The term "edema" has been substituted for "sagging of the canal wall" by this writer. TOBEY.

Continuous Stimulation of the Labyrinth with Sustained Nystagmus.

Spiegel, E. A., M. D., and Aronson, L., M. D. (New York), Arch. of Otolaryng., 17:311 (March), 1933.

Previously Spiegel had observed reflex movements of the eyes after several hours of labyrinthine stimulation.

This study was carried out with cats under superficial ether anesthesia; cold water, hot water and galvanic current were used. The external canal and middle ear were tried.

It was found that continuous caloric stimulation of the labyrinth produced nystagmoid movements of the eye, for several hours, but interrupted by short periods of rest. Spontaneous

nystagmus following extirpation of one labyrinth could be slowed by cold stimulation of the remaining one.

The direction of this nystagmus was usually influenced by the position of the head, as long as endolymph flow was the factor, but the end-organ apparently became paralyzed in some cases and then the nystagmus was changed. TOBEY.

MISCELLANEOUS.

On the Structure of the Subglottic Region of Man and Some Mammals (Sulla Struttura Della Regione Ipoglattica Nell'Uomo ed in Alcuni Mammiferi).

Menniti-Ippolito, R. (Naples), Arch. Ital. di Otol., Rino. e. Laryng., 44: 33 (January), 1933.

The author reviews the literature concerning this region briefly, but to the point, quoting numerous researchers. He stresses the work of Nardi, who has brought to light the most important structures in the subglottic area—that is, the glands appear at the anterior portion as early as the 3 months fetus, and at the age of 10 or 15 years they form four distinct groups, the anterior being the largest; the elastic fibers are arranged anteriorly in large bundles in the form of a fan surrounding the glands; posteriorly they are thin and form a network. These fibers are separated into two distinct layers, the deep one thicker and below the glands, the superficial one thinner and situated between the glands and the submucosa.

He then reports his personal investigations, beginning where Nardi left off, as to why neoplasms develop more frequently in the anterior portion of the subglottic space, and compares all these structures in man and animals of various ages, presenting five instructive microphotographs.

Finally he concludes that the anterior portion of the subglottic region has the distinct characteristics to facilitate the development of neoplasms; that the glands and elastic fibers begin to develop from the early months of intrauterine life and acquire full development up to the age of 15, remaining so up to the age of 50, and after that they manifest retrograde changes as old age advances, and that in the mammals the anterior portion of the subglottic region is the most important. SCIARRETTA.

The Need of a Course in Medical Genetics in the Medical Curriculum.
Macklin, M. T., Edinburgh Medical Journal, 40:20 (January), 1933.

The author discusses the unique position which the medical practitioner holds with respect to eugenics and that the education of the medical profession in the science of medical genetics is absolutely essential for the success of the eugenic program. The attitude of the profession as a whole is not as co-operative as it should be, for their attitude depends upon their knowledge of the subject, and this they gain almost entirely when they are medical students; only a few practitioners ever acquire this information after graduation. Medical students are taught very little of inheritance in disease and practically their only training comes in the premedical courses. Medical genetics should be taught in the last year of the medical curriculum when the students have become familiar with the phenomena of disease. It is essential that physicians publish their records of inherited disease, so that the student of inheritance may have the data which can only be gathered by the medical profession. GOLDSMITH AND MCLEOD.

Peripheral Neuritis: The Morrison Lectures, 1932, Delivered Before the Royal College of Physicians of Edinburgh, May, 1932.
Collier, J., Edinburgh Medical Journal, 39:672 (November), 1932.

Lecture 2. The author presents evidence that in peripheral neuritis the muscular part of the spinal reflex arc may be primarily affected, and that essential lesions of the muscle itself play an important part in the symptomatology of some varieties. For peripheral neuritis to be of the painful form there must be either an interstitial lesion outside of and irritating the sensory nerve terminals or there must be an interstitial lesion of the nerve trunk. There is no evidence that degeneration of the peripheral nerve elements or that pressure upon the nerve fibers in the nerve trunks ever causes pain. The author has seen the fifth nerve utterly destroyed by pressure of a secondary growth on many occasions without the patient having had the least discomfort in that region, and has made similar observations on other sensory-containing nerve trunks of the body.

There are filter-passing viruses in certain diseases, such as poliomyelitis, which gain access to the nervous system by a purely axonic route and usually enter by the olfactory nerve terminals

in the upper meatus of the nose. They may then spread to every nerve filament in the body. Virus infections may live within the nervous system for long periods, with recurrences or increase of symptoms very long after the original infection, as in the cases of lethargic encephalitis and herpes zoster.

Facial paralysis of Bell's type may be a herpes zoster proper. In some cases in which there is no meatal vesicle, zoster may be present in some other region of the body, and facial paralysis, without local herpetic signs, can result from zoster infection. Two interesting cases were reported. The first case was a left-sided facial paralysis with a very sore throat. The palate was completely paralyzed on the left side, and on the front of the soft palate on the same side there was a crop of herpes zoster. The second case was a left-sided facial palsy with well marked meatal vesicle. There was a complete unilateral palate palsy, and a crop of herpes zoster on the front of the paralyzed half of the palate, but this was on the opposite side to the facial palsy and therefore could have had nothing to do with the facial paralysis. The explanation given is that in both cases there were multiple herpetic lesions both in the geniculate ganglion of the facial nerve and in Meckel's ganglion.

Peripheral neuritis occurring in infections, poisoning and epidemics is discussed. A true cause is yet to be found for the majority of the present day cases of peripheral neuritis.

GOLDSMITH AND MCLEOD.

Pulmonary Disinfection and Immunization.

Kolmer, John A. (Philadelphia), Trans. Am. Laryng. Assoc., 1933.

It would appear that intrabronchial instillations of bactericidal and spirocheticidal chemical agents as well as of bacteriophages and antiviruses may constitute hopeful measures in the treatment of chronic bronchitis, bronchiectasis and abscess of the lung in addition to drainage of infected sinuses, bronchoscopic and postural drainage and other measures. A large number of chemical agents, vaccines, antiviruses, etc., have been given to rabbits intrabronchially to determine toxicity, tolerance and effects on the tissues in establishing the basic groundwork of this therapy. A summary of the results was given with recommendations.

IMPERATORI.

Permanent Enlargement of the Lips and Face, Secondary to Recurring Swellings and Associated with Facial Paralysis: A Clinical Entity.

New, Gordon B. (Rochester, Minn.), Trans. Am. Laryng. Assoc., 1933.

In the last twenty-two years, sixty-seven patients presenting chronic, noninflammatory, permanent enlargement of the lips and face have been observed in the Mayo Clinic. The condition has followed repeated swellings, and has been associated with facial paralysis in thirteen cases. The condition comes on suddenly in a manner similar to angioneurotic edema, and then with the recurrence of attacks there is increasing enlargement. The condition is apparently a clinical entity, but only slight mention of it has been found in the literature. These patients have been treated by the use of injections of hot water and radium. When there is no tendency for the swellings to recur, the superfluous areas of tissue of the lips and cheeks are excised. The results of treatment of the condition have been satisfactory. IMPERATORI.

The Influence of Nasal Stimulation on the Pressure of the Cerebrospinal Fluid (L'Influenza Degli Stimoli Nasali Sulla Pressione del Liquido Cerebrospinale).

Carnevale-Ricci, F. (Milan), Arch. Ital. di Otol., Rino. e. Laryng., 44:1 (January), 1933.

The author has made experiments, on dogs, under local and light general chloral anesthesia. He inserted a cannula into the atlo-occipital cisterna and then stimulated the nasal mucosa of the various animals, some by a sharp or a rough instrument, others by a hot needle or ether, ammonia or chloroform, and the results of the stimulation were registered on a tracing paper.

With graphs he registered also the respiratory movements and noticed that the variation of respirations and cerebrospinal fluid pressure varied simultaneously, and that the mechanical stimulation caused very little alteration, while the reaction to thermic and chemical stimulation was very marked.

Later, to test whether these changes of pressure in the cerebrospinal fluid were influenced by respiratory movements, he produced a temporary asphyxia in the animal and then stimulated the nasal mucosa, but no changes occurred in the spinal fluid reading. Therefore he concluded that the pressure of the cerebrospinal fluid is changed by the respiratory pressure.

SCIARRETTA.

A Note on the Pathologic Museum.

Davies, E. D., and Cawthorne, T. (London), Otolaryngologic Section of the British Medical Association Centenary Meeting, July, 1932. Journal of Laryng. and Otol., 47:756 (November), 1932.

The increased interest in pathology of the ear, nose and throat during recent years has improved the scientific reputation of the specialty and has raised its status.

The specimens of the museum were divided into two main groups: specimens of abnormalities of the trachea, bronchi and lungs to illustrate bronchoscopy, and a collection of over 100 specimens of nasal sinus suppuration. In the first group there were twenty-two specimens of bronchi and lungs with foreign bodies in situ. They were all more than 20 years old and before efficient bronchoscopes were in use. These specimens confirmed an already recorded fact that the pathologic lesions depend on a number of details, such as the character of the foreign body, its position, the age of the patient and the mechanical action of the offender. There were a number of specimens of growths of the lungs which involved the trachea and bronchi, and also some inflammatory diseases of the trachea, mediastinal glands and lungs which could be recognized by the bronchoscope.

A splendid collection of nasal sinus suppuration specimens was shown and the method of obtaining them intact is given. Some excellent specimens of the mucosa of the antrum, to demonstrate the pathologic changes resulting from latent chronic inflammation, were exhibited. GOLDSMITH AND MCLEOD.

Peripheral Neuritis: The Morrison Lecture, 1932, Delivered Before the Royal College of Physicians of Edinburgh, May, 1932.

Collier, J., Edinburgh Medical Journal, 39:697 (December), 1932.

Lecture 3. Disease of the spinal cord, such as syringomyelia, in which a mysterious peripheral neuritis seems sometimes to occur, are discussed. In diabetes mellitus there are four associated conditions of neuritis, each of which seems quite different to and separate from the others. One variety of neuritis occurring in diabetes is paralysis of the oculomotor nerves, commonly of the sixth, but sometimes of the third, and rarely of the fourth. Most cases of ocular paralysis in diabetes recover perfectly and some of them very quickly upon dieting alone or with the use of insulin.

In the author's experience diphtherial infection of the chronic carrier order was the most common cause of recurring neuritis, and in several cases the Klebs-Loeffler bacillus of diphtheria was found in the antrum or other accessory cavities of the nose. Diphtheria palsy usually affects the ciliary muscle in the eye alone, yet it may cause paralysis of one or more ocular nerve trunks. It may cause an irregular nuclear ophthalmoplegia. The sensory elements are usually completely avoided with the exception of the palate and pharynx.

Pathologic changes in the cerebrospinal fluid in peripheral and interstitial neuritis are discussed.

A number of unusual cases of neuritis and chemical experiences are given in the article. GOLDSMITH AND MCLEOD.

Abnormalities of the Upper Respiratory Tract and Minor Respiratory Diseases.*

Doull, J. A., Von Volkenburgh, V. A., Herman, N. B., and Gafafer, W. M. (Baltimore), Amer. Jour. Hyg., 17:743-760, May, 1933.

There is no unanimity of opinion upon the question of the relationship of abnormalities of the upper respiratory tract to minor respiratory diseases. It is not infrequently stated that these diseases are more frequent and more severe in persons having some objectively demonstrable pathologic condition of the upper respiratory tract than in normal individuals. One of the opportunities afforded by the establishment of the John J. Abel Fund for Research on the Common Cold at the Johns Hopkins University was the intensive study of the possible importance of these abnormalities in the susceptibility to this group of diseases.

During the academic years 1928-1929 and 1929-1930 approximately 2,500 attacks suffered by about 500 persons were recorded and studied. The persons were members of either of two groups: (1) chiefly students of the School of Medicine, and the School of Hygiene and Public Health, and (2) families resident in various sections of Baltimore, and for present purposes divided into two age groups, namely, less than 15 years, and 15 years and over. At entrance to the study, when free of upper respiratory disease,

*From the Clinical Laboratory of the John J. Abel Fund for Research on the Common Cold, and the Departments of Epidemiology and of Biostatistics (Paper No. 183) of the School of Hygiene and Public Health, the Johns Hopkins University.

each person was examined in the Otolaryngologic Clinic of the Johns Hopkins Hospital. The present report required the removal from the otolaryngologic examination records of data concerning the following characteristics of each person:

1. Palatine Tonsils.—Inflammation; hypertrophy, or removal. Removal was classified as complete only if no remnants or remnants insignificant in amount were observed.

2. Adenoids. Inflammation; size of mass, whether large, moderate or small (normal); removal.

3. Breathing Space.—A record was made of each side of the nose. The chief defects causing obstruction were septal spur, hypertrophy of the mucous membrane covering the middle or inferior turbinates, and, in a few instances, polyps.

4. Mucous Membrane of the Turbinates.—Emphasis was placed on the presence or absence of thickened and polypoid conditions of the mucous membrane of the turbinates since these may indicate a chronic ethmoid infection.

5. Maxillary Sinuses.—Clear or cloudy on roentgenography.

A person was classified as normal in respect of the upper respiratory tract when all of the following characteristics were present; otherwise he was regarded as abnormal:

1. Tonsils completely removed, or normal in appearance if present in whole or in part.
2. Adenoids normal in appearance or completely removed.
3. Good breathing space on both sides.
4. Middle and inferior turbinates normal in appearance.
5. Maxillary sinuses clear on roentgenography.

The results of the study may be conveniently summarized thus:

1. Taken as a group, persons exhibiting one or more abnormalities were found not to suffer a greater frequency of attack than normal persons. Those with abnormal tonsils or adenoids, however, had a questionably higher rate than normal persons, and persons on whom a corrective operation had been performed suffered higher rates than either of these classes. This latter result may be due to selection of unusually susceptible persons for operation.

2. With regard to the relative severity of attack in normals and abnormals, respectively, it was found that so far as confinement to bed and general aching may be taken as criteria, attacks among abnormals were of approximately the same severity as those among normals. The relationship of fever to the presence of abnormality is a more complicated question. Attacks in those with diseased tonsils were no more frequently accompanied by

fever than attacks in normal persons. However, it is of interest that in the student group those with this abnormality showed a quicker febrile response. Also young children with excessive adenoid tissue showed a slightly greater tendency to fever than children normal in this respect. It is of interest that the study points to an apparently greater frequency of productive cough in those with abnormal turbinates and in those with abnormal maxillary sinuses than in normal persons. Among children the average duration of attack was longer for those with each class of defect than for those who were recorded as normal.

3. Persons with abnormality, whether of tonsils, adenoids, breathing space, turbinates or maxillary sinuses, are more likely than normal persons to have nasal discharge on the first day of illness; and in those with the particular defect, namely, abnormality of tonsils, it was found that sore throat was no more frequently present on the first day of attack than in normal persons.

AUTHOR'S ABSTRACT.

Radium Treatment of Esophageal Cancer (Le Curiethérapie des cancers de l'oesophage).

Ducuing, Dr. L. (Toulouse), Rev. de Lar., 54:470 (April), 1933.

Reporting on fifty-five cases treated, the author prefers radium to deep X-ray therapy. The growth, if near the cardia, may be accurately mapped by getting some radiopaque broth into the stomach, then putting the patient on the screen table in the Trendelenburg position, while he swallows a few spoonsful of the mixture. That in the stomach will flow back, and the growth will be outlined between the two opaque accumulations.

Ducuing advises against gastrostomy save in very well nourished patients, and considers treatment useless if the growth is more than 5 cm. long, or in the presence of mediastinitis, perioesophageal gland masses or other metastases; also in the presence of lung involvement or severe cachexia.

After training the patient to swallow a thread, two or three 5 mgm. radium tubes separated from each other by 1 cm. of cotton in the end of a 22 or 24 rubber catheter are introduced to the level previously ascertained. A total of 260 to 3,000 hours of exposure, in sessions of 24 to 48 hours, is required.

Crossfiring the neck with radium installed in a wax-sawdust

collar is described, but not approved unless the catheter method is impracticable. Prognosis is bad in every case, although many months of palliation may be attained. Such tumors are not especially radiosensitive, and if the cancer is destroyed the esophagus itself suffers and the patient dies from mediastinal or pulmonary complications. FENTON.

The Radicular Cyst Which Invades the Bones of the Face.

Alden, Arthur M. (St. Louis), Trans. Am. Laryng. Assoc., 1933.

There are two types of dental cysts which are of interest to the rhinologist because of the fact that they often invade the bony portion of the maxilla: the follicular cyst which develops from and around the crown portion of a malposed tooth, and which always includes one or more teeth in its periphery, and the radicular cyst which originates from the diseased apical portion of a tooth. The latter type of cyst quite commonly extends upward into the maxillary sinus and is often mistaken for antral suppuration. The author described the histopathology of the radicular cyst and the means by which it so readily invades the bones of the face; the differential diagnosis between a radicular cyst which occupies the bony cavity of the maxillary sinus and maxillary sinusitis proper, and the methods of surgical treatment. Slides were shown, illustrating the histology of the condition. IMPERATORI.

Mucocele as a Cause of Proptosis: Detail of Six Cases.

Chamberlin, William B. (Cleveland), Trans. Am. Laryng. Assoc., 1933.

According to Vail, the term "mucocele" was first suggested by Rollet in 1896. The term rather is applied to a condition in which there is an accumulation and retention of a thick, mucoid, gelatinous secretion within a sinus. This is due to an obstruction of its outlet and may be caused by: (1) trauma; (2) an acute inflammatory process with consequent swelling; (3) pressure on the outlet from a new growth, such as an osteoma; (4) a chronic inflammation within a sinus where the bacteria have lost their virulence, and the pus has become sterile. The process is of slow growth and is long continued, resulting in thinning of the bony walls of the sinus and displacement of the eye, outward, downward and usually forward. A report is made of six cases in patients ranging in age from 12 to 43 years. IMPERATORI.

Congenital Fistulae of the Neck (Las fistulas congenitas del cuello).

Bergara, Drs. R. and C. (Buenos Aires), An. de O. R. L. Uruguay, 2:117 (August), 1932.

Case report of a woman of 21, with a 1 mm. fistula discharging seropurulent fluid in front of the sternomastoid at the level of the hyoid bone, opening above into the outer third of the external auditory meatus. There was a history of several large swellings along this course with discharge of considerable pus.

The fistula was obliterated, after preliminary syringing with 2 per cent novocain, by repeated injections of a modified Morestin solution (āā formalin sol., tricresol, alcohol, glycerin, 5 cc.; novocain sol. 2 per cent, 20 cc. diluted 1:4 with water). The skin of the neck and of the meatus must be protected by vaselin.

This is the ninth case so far reported of this particular sort of fistula. FENTON.

Carcinoma of the Lung and Bronchus: Treatment with Radon Implantations and Diathermy.

Kernan, John D., M. D. (New York), Arch. of Otolaryng., 17:457 (April), 1933.

In this article ten cases are reported in detail with X-ray pictures and pathologic slides shown.

The subject is discussed as regards symptomatology, early diagnosis, treatment and prognosis. Of sixty cases bronchoscoped only five were early enough to offer any chance of cure.

Early symptoms may be cerebral, gastric, debility or pointing to other distant organs.

The author usually found bronchiectasis or abscess below the point of the new growth.

One case suggested to Dr. Kernan that histologic carcinoma and the disease carcinoma are not the same, in every instance.

Discussion by Drs. Pancoast, Lejeune, Taquino, Walker, Kernan and Arbuckle ended the paper. TOBEY.

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